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Winning Publication of The Medical Society of Virginia

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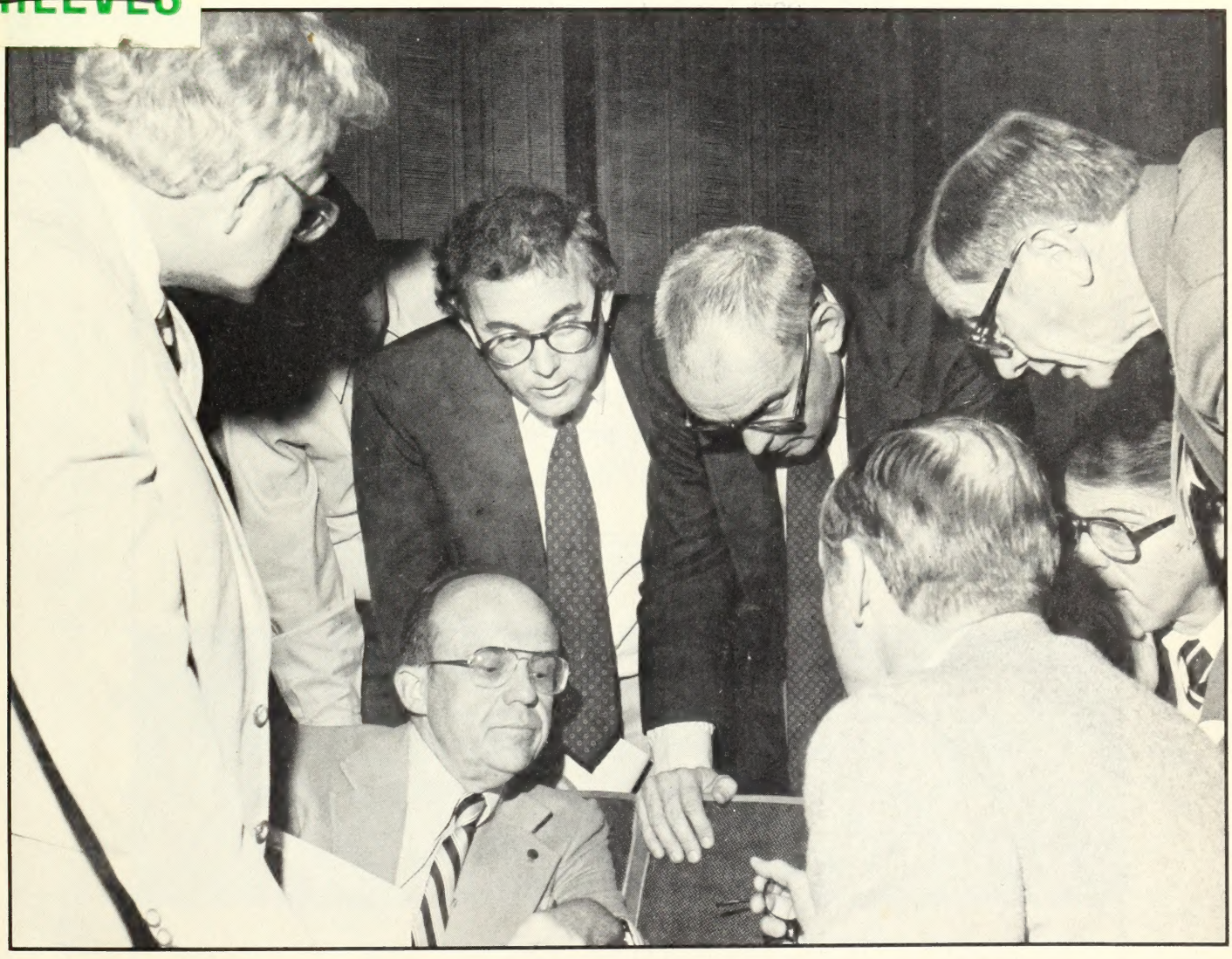

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Volume 110 January 1983 Number

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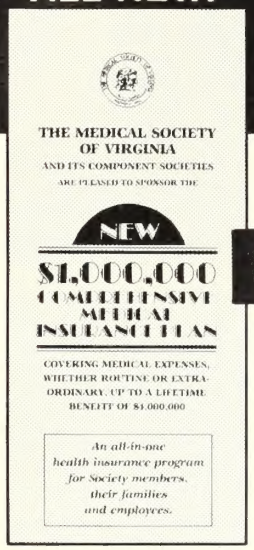

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VIRGINIA MEDICAL

January 1983, Volume 110, Number 1

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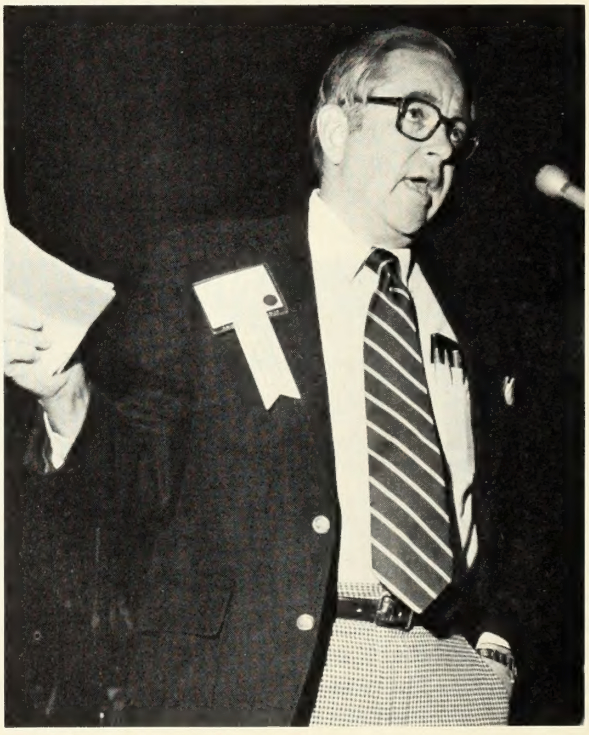
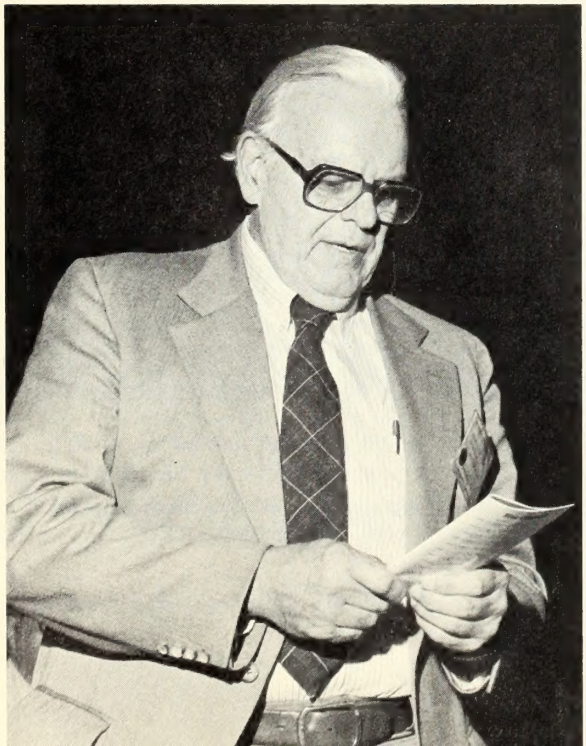
The 5th District caucuses.

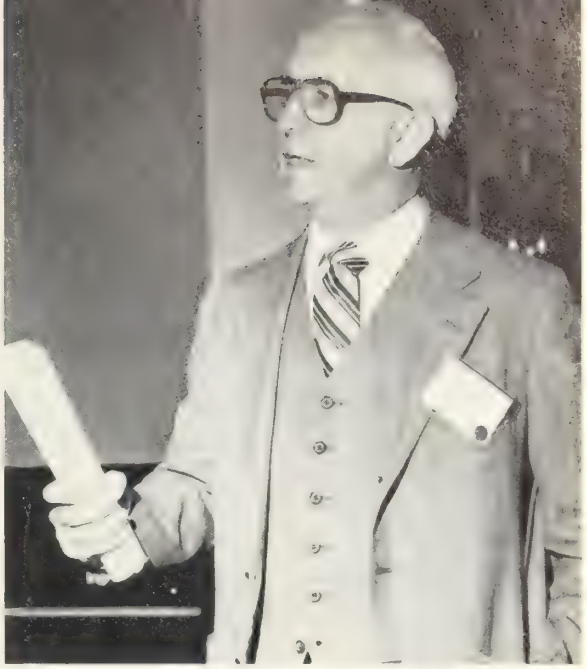
Medical Society of Virginia members meet in Williamsburg

Dr. Duncan S. Owen, Jr., Richmond



Dr. William S. Terry, Portsmouth





Dr. Robert L. A. Keeley, Roanoke

Gov. Charles S. Robb, VaMPAC speaker. The Governor described the Medicaid-financing dilemma brought about by budget cuts and asked the help of physicians in holding down costs.

Dr. James E. Nevin III, Danville

Dr. Ira J. Green, Alexandria

Dr. John A. Owen, Jr., Charlottesville
For Delegate Owen as author, see page 59.

Dr. William L. Rich III, Fairfax

Dr. William D. Mayer, Norfolk

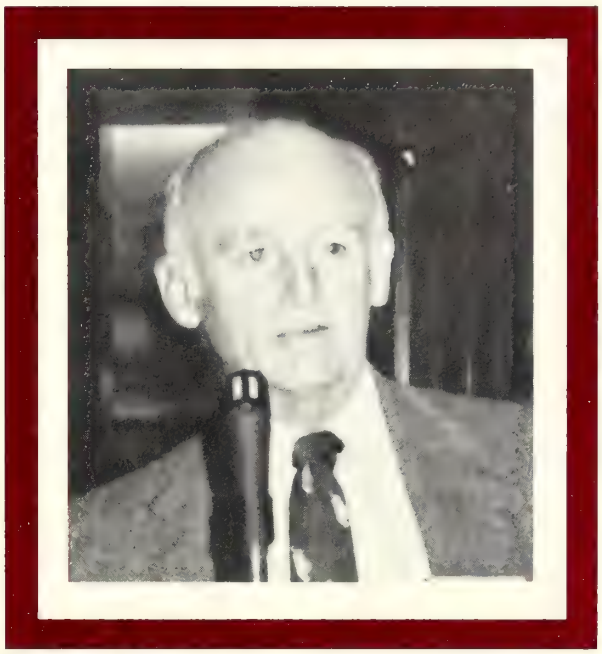
Dr. Michael A. Puzak, Arlington

Dr. Robert T. Mosby, Jr., Virginia Beach

Dr. Richard J. Hart,. Jr., Fairfax

The 1st District caucuses.

Medical Society of Virginia Officers 1983



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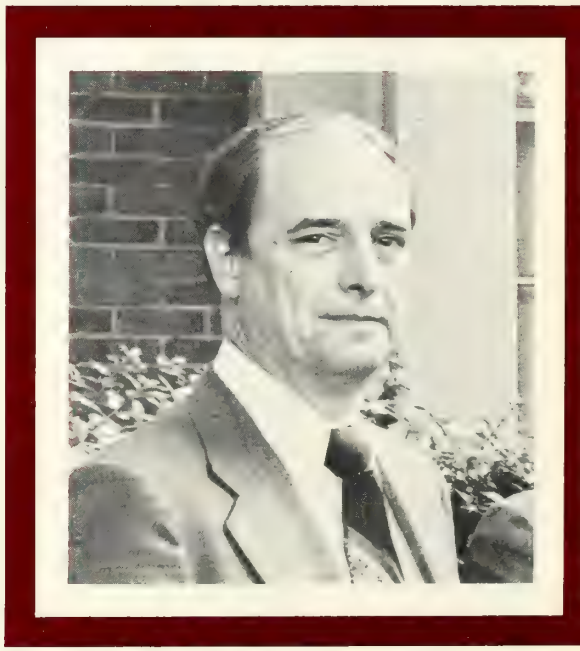
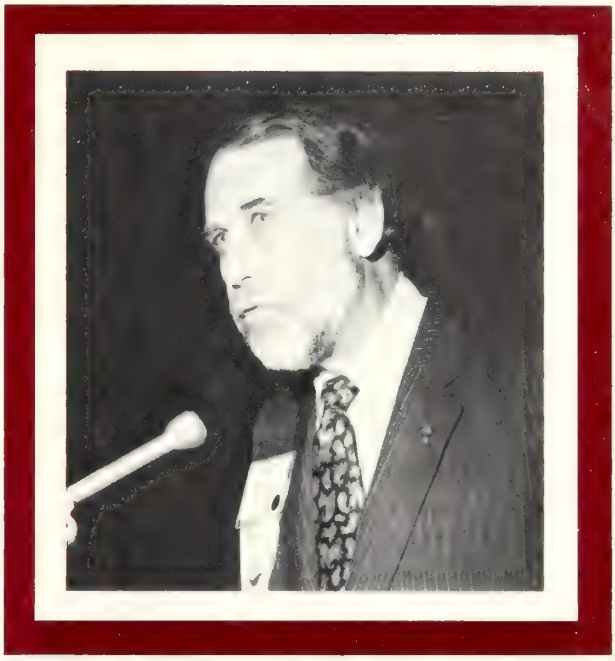
C. Barrie Cook, MD, Fairfax

1st Vice President
Harry C. Kuykendall, MD, Alexandria



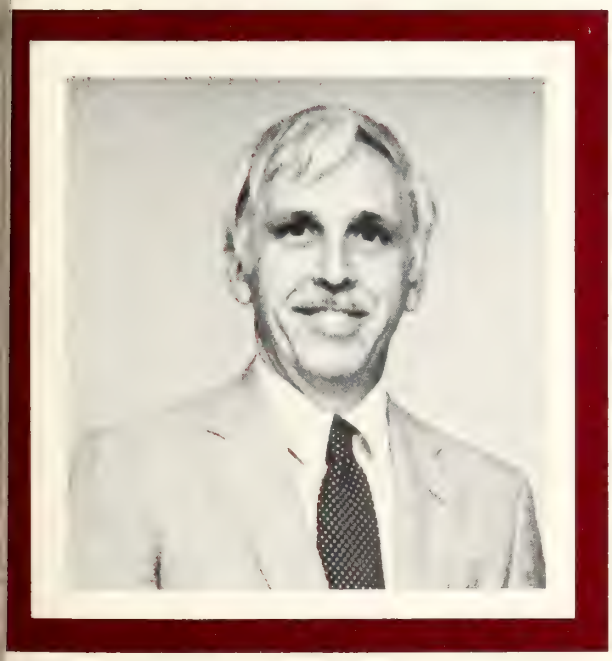
2nd Vice President
Frederick K. McCune, MD, Virginia Beach

New President Harold L. Williams, MD, Newport News (right), at the VaMPAC banquet with (left) Gov. Charles S. Robb and (center) VaMPAC Chairman Percy Wootton, MD, Richmond.



Speaker of the House
Richard L. Fields, MD, Fairfax

Vice Speaker
William H. Barney, MD, Lynchburg



3rd Vice President
H. Dale Sponaugle, MD, Portsmouth

Photographs in this section by Thomas L. Williams, Denise Doverspike, William Abourjilie and Melissa Grimes-Guy.

THE HOUSE OF DELEGATES

Medical Society of Virginia policy and programs begin in the Society's committees and component medical societies, who once yearly, at the annual meeting, present to the House of Delegates proposals for action. The Speaker of the House places these proposals in the hands of reference committees; they conduct open debate on the issues and then formulate for the House their recommendations for disposition. The assembled House then votes to accept or reject the recommendations.

Summarized here are highlights of the House of Delegates' actions at the 1982 annual meeting.

At its session on November 13, 1982, in Williamsburg, Medical Society of Virginia delegates

• **Called** for the use of contracts between local health departments and providers of maternity and newborn services. **Supported** the development of regulations requiring birthing centers to be physically connected with an acute care center and supervised by the involved hospital.

• **Endorsed** the concept of cost-sharing in all health insurance programs (deductibles and copayments with provisions for annual stop-loss). **Advocated** replacing cost-plus reimbursement of hospital charges and a study of alternative mechanisms. **Endorsed** health care plans that encourage outpatient surgery while preserving quality of care and safety. **Endorsed** education programs encouraging all citizens to retain primary physicians. **Endorsed** the concept of local peer review. **Reaffirmed** the concept of leaders of medicine and business discussing problems of mutual concern.

• **Asked** for formation of a "joint advisory committee" on sports medicine made up of representatives from the Virginia Department of Education, Virginia High School League, Virginia Physician Therapy Association and Virginia Athletic Trainers Association.

• **Recommended** that The Medical Society of Virginia and the Virginia State Bar and judiciary in local and regional areas work together to establish guidelines, rules or standing orders of courts with reference to fees paid for expert testimony by physicians.

• **Voted** in favor of establishing a multi-specialty emergency medical committee to work with the Virginia Chapter of the American College of Emergency Physicians.

• **Supported** increased efforts to 1) collect on overdue government loans for medical study and 2) counsel student and resident borrowers on obligations pertaining to such indebtedness.

• **Directed** The Medical Society of Virginia to seek a contract with the Department of Health and Human Services as a Medicare peer review organization under the Peer Review Improvement Act of 1982, known as the Durenberger Bill.

• **Did not adopt** a resolution supporting a continuing moratorium on uranium exploration, milling and mining. The reference committee suggested this rejection, on the grounds that a continuing moratorium would indefinitely delay resolution of safety issues facing the uranium industry but asked the Society to continue monitoring developments in this industry through its representation on appropriate advisory and regulatory boards.

• **Voted** to establish a program effective January 1, 1983, to assist in the rehabilitation and recovery of physicians with physical, mental or substance abuse problems adversely affecting their ability to practice medicine. **Adopted** a new Section 5 to Article IXA of the Society's Bylaws that establishes the Physicians' Health and Effectiveness Committee as a standing committee of the Society.

• **Adopted** a recommendation implementing a utilization management program in conjunction with the Society's group Blue Cross-Blue Shield plan.

• **Directed** the Long-Range Planning Committee to 1) consider the establishment of an insurance brokerage to administer various types of insurance programs for Medical Society of Virginia members; 2) study the establishment of a subsidiary corporation to provide administrative and secretarial services to specialty organizations; 3) review the space problem at Medical Society of Virginia headquarters and formulate a plan for expansion.

• **Endorsed** continuance of the Negotiations Committee and the provision of opportunities for formal negotiations training for interested members.

• **Asked** the Bylaws Committee to study an approach for selecting councilors and vice councilors that does not use the state congressional districts.

• **Accorded** H. C. Alexander III, MD, President of The Medical Society of Virginia 1981-1982, the status of honorary active membership. For Dr. Alexander's address to the delegates, see page 60.

• **Called** for the Membership Committee to solicit AMA members with the help of the Society's AMA delegation.

• **Agreed** to submit all of the proposed changes in the Society's Articles of Incorporation to a vote of the voting members. **Adopted** bylaw amendments to provide for specialty representation in the House, shift the provision regarding voting rights of members from the Articles of Incorporation to the Bylaws, and redefine the functions of the Insurance Committee to make the applicable bylaw consistent with the current functions of the committee.

At the meeting of voting members, amendments of the Articles of Incorporation were adopted which 1) provide for representation of specialty groups; 2) give full membership rights to active student members; 3) recognize the Eastern Virginia Student Medical Society as a component student society; 4) shift the provision for voting rights of members from the Articles of Incorporation to the Bylaws; 5) limit active membership in The Medical Society of Virginia to active members of component societies; 6) clarify the Society's position on exemption from dues. The voting members rejected an amendment

Delegates set legislative positions

The House of Delegates took these positions on legislation scheduled for consideration by Virginia's General Assembly at its 1983 session beginning this month:

• **Oppose** amendment of the Virginia Code to prohibit physicians from transmitting orders for hospitalized patients through employees.

• **Support** repeal of the present law requiring premarital serologic tests.

• **Oppose** efforts to reduce or eliminate loan funds now available to Virginia medical students and **support** new programs to provide such financial assistance.

• **Support** enforcement by the Virginia State Board of Medicine of licensure statutes restricting "limited practitioners" to the scope of their authorized practices.

• **Support** stiffer penalties for drunk driving.

• **Support** repeal of the National Health Planning Resources Development Act (Public Law 93-641).

• **Oppose** any attempt to change the statute of limitations for personal injury actions to a discovery rule.

• **Oppose** the adoption of the comparative negligence doctrine.

• **Oppose** elimination of the $750,000 cap on medical malpractice awards.

• **Oppose** any special statutory requirement of informed consent for treatment of breast cancer.

• **Oppose** any statute that would require licensure of respiratory or occupational therapists by the Virginia State Board of Medicine.

• **Oppose** further cuts in the State's Medicaid budget unless it is clear that such cuts will not adversely affect quality of care for the poor.

• **Oppose** legislation to authorize the use of diagnostic drugs by optometrists unless the legislation also requires referral to a physician of any patient with an abnormality that customarily requires medical evaluation or treatment.

• **Support** legislation that would provide civil immunity for members of The Medical Society of Virginia's Physicians' Health and Effectiveness Committee and the program's intervenors and investigators as long as they act in good faith.

• **Do not oppose** statutory confirmation of the right of the terminally ill to reject further treatment as long as there is no obligation on the physician's part to comply with the patient's request and appropriate immunity is provided if the physician complies.

• **Oppose** any increase in the type of services that the Virginia Code requires in health insurance policies.

• **Support** any statutory changes that may be necessary to confirm that the existing sales tax exemption for sales to patients of orthopedic and other devices also applies to purchase by physicians for ultimate resale to the patient.

He plays neither tennis nor golf, so Dr. Henry S. Campell of Martinsville brought his motorcycle to the annual meeting and, when his duties as a delegate allowed, cruised through the Williamsburg countryside. Here he regales Dr. Francis D. Andres, Annandale, with his easy-rider role. For Dr. Campell as author, see page 58.

relating to direct membership in the Society for interns and residents.

• Approved the following budget for the fiscal year ending September 30, 1983:

EXPENSES:	**Proposed**
Salaries, including continuing medical education	$273,000
Printing and mailing services—membership	2,500
Stationery and supplies	9,500
Office equipment: repairs and replacements	7,500
Building maintenance and repairs	31,000
Telephone	7,500
Aquisition of new telephone equipment	16,830
Postage	8,500
Convention expenses	20,000
Council and committee expenses	14,000
Travel expense:	
Delegates to AMA	18,000
Executive Vice President and staff	6,500
Virginia Medical	110,000
Legal expense	75,000
Walter Reed Commission	1,500
Auxiliary	2,500
Membership dues (affiliated organizations)	1,100
Editor, Virginia Medical	2,400
VaMPAC (educational fund)	50,000
Newsletter	3,500
Presidential expense allowance	15,000
Presidential expense account	12,500
Employee benefits:	
Retirement fund	65,000
Payroll taxes	19,000
Blue Cross/Blue Shield, life and disability insurance	14,000
Legislative expense	11,000
Continuing medical education	6,000
Miscellaneous	3,000
Special Appropriations:	
Virginia Council on Health and Medical Care	12,000
AMA-ERF	1,000
Rural health	1,500
Scholarships administered by The Medical Society of Virginia:	
MCV	2,000
UVa	2,000
EVMS	2,000
Miscellaneous AMA	5,500
Miscellaneous appropriations, includes insurance premiums on premises and Workman's Compensation	2,500
Consultants:	
Legislative, public relations and actuarial services	75,000
Auditor	3,000
Student medical societies:	
MCV	2,500
UVa	2,500
EVMS	2,500
Computer operation and maintenance	7,000
Physicians' Health and Effectiveness Program	10,000
TOTAL EXPENSES	$937,330

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AMA Delegates	**Michael A. Puzak, MD,** *Arlington* **Carl E. Stark, MD,** *Wytheville* **W. Leonard Weyl, MD,** *Arlington* **F. Ashton Carmines, MD,** *Newport News* **John A. Martin, MD,** *Roanoke* **Raymond S. Brown, MD,** *Gloucester*
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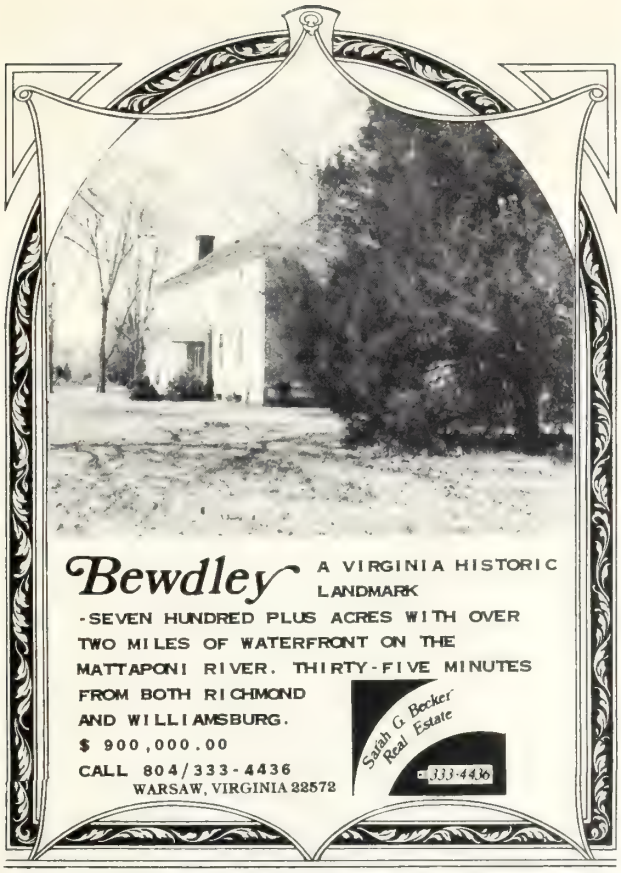
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LETTERS

Disability Review Misunderstood, Creating Need for Physicians

The Disability Determination Services is a division of the Virginia Department of Rehabilitative Services. Its mission is to provide fair and timely decisions on the claims by Virginians for disability benefits under the Social Security Act. Such decisions are made in accordance with the provisions of this Act.

In 1980 Congress amended the Social Security Act to require the periodic review of nealry all Social Security disability beneficiaries every three years. This requirement has made it more difficult for the Disability Determination Services to fulfill its mission. Of particular concern is that this requirement for periodic review has had an adverse effect on the Service's relations with Virginia's medical community.

Periodic review has created confusion for the treating physician. He does not understand why he is being requested to provide evidence of disability on his patient, who, he thought, was already receiving Social Security benefits as permanently and totally disabled. In some instances, this patient may have been receiving benefits for a decade or more. Even when the procedure is explained, the physician may feel that the responsibility will fall on him if the patient's benefits are terminated. And, finally, his already heavy burden of paperwork is being increased by these requests for further reports on his patients.

In addition, as the result of periodic review pending caseloads have reached record levels and are still rising. Many of the persons who are receiving disability benefits have had little or no medical treatment since the establishment of the onset of payments. This situation has caused an increase in the scheduling of consulting examinations for current medical information, and the tremendous volume of examinations has severely taxed physicians providing these consulting examination services. Because of the possibility of cessation of benefits, some consulting physicians understandably are reluctant to perform disability evaluations.

As a result of the impact of periodic review, the

Disability Determination Services urgently needs the understanding and assistance of the medical community in the following:

- Periodic review will be affecting almost all recipients of Social Security disability benefits.
- The Disability Determination Services is implementing procedures to minimize the burden of its requests for medical evidence on the treating physician.
- Neither the treating nor the consulting physician is responsible for any possible cessation of an individual's benefits. The physician is requested to provide the medical evidence, supported by clinical, laboratory and X-ray findings, upon which the impairment can be evaluated. The effect of the impairment on an individual's ability to work and the decision as to whether or not the impairment constitutes a disability is an administrative decision, according to Social Security Regulations.
- The Disability Determination Services has immediate and pressing need for board-certified specialists to perform consulting examinations, especially in the areas of internal medicine, family practice, orthopedics, neurology and psychiatry.

Despite the problems encountered, the 1980 legislation is an important amendment to the Social Security Act. It is essential that beneficiaries ineligible for compensation be removed from the rolls. At the same time, those individuals who are indeed disabled deserve fair and timely review of their disability in order to keep their monthly benefits intact.

Physicians in Virginia have long been noted for the deep concern they have evidenced and the excellent services they have provided for disabled citizens of the Commonwealth. The need is greater than ever for the Disability Determination Services to have the continued cooperation and assistance of Virginia's physicians. If you are willing to perform consulting examinations or if you desire further information about the Services or the periodic review process, please call me station-to-station collect at (804) 257-0186.

Reno R. Porter, MD

Chief Medical Consultant
Department of Rehabilitative Services
PO Box 11045, Richmond VA 23230-1045

A Brief Summary
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INDICATIONS AND USAGE: Melfiat® 105 (phendimetrazine tartrate) is indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class (See CLINICAL PHARMACOLOGY) should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, symptomatic cardiovascular disease, moderate to severe hypertension, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors (hypertensive crises may result).

WARNINGS: Tolerance to the anorectic effect usually develops within a few weeks. When this occurs, the recommended dose should be discontinued. Phendimetrazine tartrate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly.

Drug Dependence: Phendimetrazine tartrate is related chemically and pharmacologically to the amphetamines. Amphetamines and related stimulant drugs have been extensively abused, and the possibility of abuse of phendimetrazine tartrate should be kept in mind when evaluating the desirability of including a drug as part of a weight-reduction program. Abuse of amphetamines and related drugs may be associated with intense psychological dependence and severe social dysfunction. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high-dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG, manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxication is psychosis, often clinically indistinguishable from schizophrenia.

USAGE IN PREGNANCY: The safety of phendimetrazine tartrate in pregnancy and lactation has not been established. Therefore, phendimetrazine tartrate should not be taken by women who are or may become pregnant.

USAGE IN CHILDREN: Phendimetrazine tartrate is not recommended for use in children under 12 years of age.

PRECAUTION: Caution is to be exercised in prescribing phendimetrazine tartrate for patients with even mild hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of phendimetrazine tartrate and the concomitant dietary regimen. Phendimetrazine tartrate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage.

ADVERSE REACTIONS: Cardiovascular: Palpitation, tachycardia, elevation of blood pressure.
Central Nervous System: Overstimulation, restlessness, dizziness, insomnia, euphoria, dysphoria, tremor, headache; rarely psychotic episodes at recommended doses.
Gastrointestinal: Dryness of the mouth, unpleasant taste, diarrhea, constipation, other gastrointestinal disturbances.
Allergic: Urticaria
Endocrine: Impotence, changes in libido

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Reference: 1. Sheu YS, Ferguson JA, Cooper JR: *Evaluation of the Abuse Liability of Diethylpropion, Phendimetrazine, and Phentermine*, unclassified document, ADAMHA, HHS. Office of Medical and Professional Affairs, NIDA, 1980, pp 10-15.

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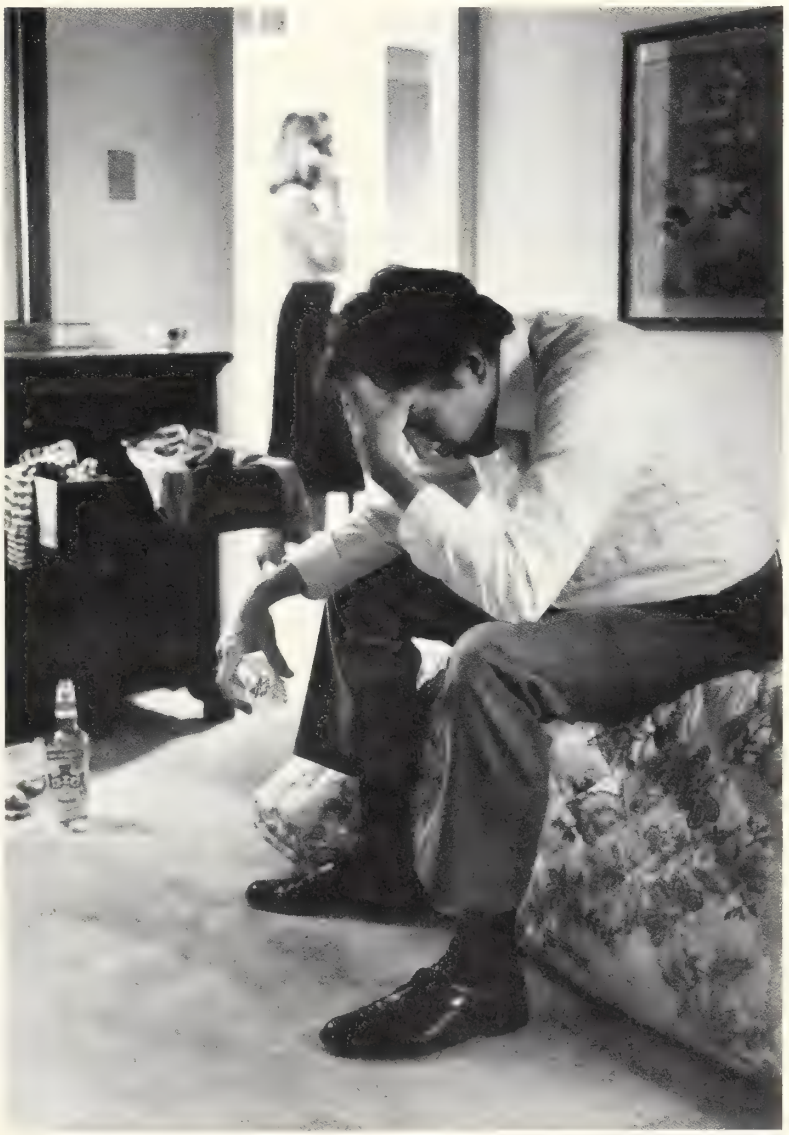

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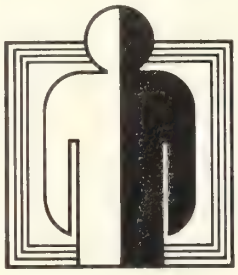

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MEETINGS ABOUT MEDICINE

January 20-23
Annual Meeting, Neurosurgical Society of the Virginias, *Hot Springs.* Robert P. Singer, MD, 7702 Parham Road, Richmond VA 23229, (804) 270-9090.

January 22
Pediatric Asthma and Allergy (Johns Hopkins), *Baltimore.* 7 hrs. credit. Fee: $100. Carlita M. Kearney, 720 Rutland Ave., Baltimore MD 21205, (301) 955-3168.

February 10-11
Trauma Care 1983 (Eastern Virginia Inter-Hospital Medical Education Committee), *Norfolk.* 12 Cat. I credits. Fee: MDs, $150; residents, $100; nurses, $80. EVIMEC, PO Box 1980, Norfolk VA 23501, (804) 446-5240.

February 10-12
Nuclear Cardiology: Methods and Applications (Johns Hopkins University), *Baltimore.* 20 Cat. I credits. Fee: $275. Susan Bavaro, Office of CME, 720 Rutland Avenue, Baltimore MD 21205.

February 11-13
Biomedical Topics in Psychiatry, Sleep Disorders and Substance Abuse (Medical College of Virginia), *Hot Springs.* 13½ Cat. I credits. Valerie Bloom, Box 48, MCV Station, Richmond VA 23298, (804) 786-8703.

February 11-13
Seventh Annual Heart-Lung Critical Care Conference (American Heart Association/American Lung Association), *Williamsburg.* Cat. I credits. Shirley Lipscomb, PO Box 7065, Richmond VA 23221, (804) 355-3295.

February 15-17
Pediatric Emergencies (Virginia Chapter, American College of Emergency Physicians), *Wintergreen.* 19 credit hrs. Fee, $150-300. Gwen E. Messler, PO Box 317, Highland Springs VA 23075, (804) 737-9433.

February 17-19
Otolaryngology and Ophthalmology Seminar (St. Francis Hospital), *Snowshoe Resort, West Virginia.* 15 credit hrs. Fee, $170. Snowshoe Seminar, St. Francis Hospital, PO Box 471, Charleston WV, (304) 348-5300.

February 18-20
Regional Meeting, American College of Physicians, *Williamsburg.* James M. Moss, MD, 1707 Osage Street, Alexandria VA 22302, (703) 998-6511.

February 21-24
Alton D. Brashear Course in Head and Neck Anatomy (Medical College of Virginia), *Richmond.* 40 credit hrs. Fee, $300. Hugo R. Seibel, MD, Box 709, MCV Station, Richmond VA 23298.

February 25
Psychobiology in the Family: The Role of Genetics in Psychiatry (Psychiatric Institute Foundation), *Washington DC.* Marie Giblin, 4460 MacArthur, NW, Washington DC 20007, (202) 467-4538.

March 6-10
Gastrointestinal Radiology: 19th Annual Post-Graduate Seminar (Medical College of Virginia), *Williamsburg.* 19½ Cat. I credits. Fee: MDs, $350; residents, $175. Sheri Rosner, Box 48, MCV Station, Richmond VA 23298.

March 9-12
Internal Medicine 1983 (University of North Carolina), *Chapel Hill, North Carolina.* Office of CME, 231 MacNider Building, Chapel Hill NC 27514.

March 17-18
Spring Meeting of the Virginia Chapter, American College of Surgeons, Hyatt House, *Richmond.* George A. Parker, MD, 8915 Norwick Road, Richmond VA 23229.

March 19
Third Annual Seminar of Cancer Researchers in Virginia (American Cancer Society, Virginia Division and Medical College of Virginia Cancer Center), *Richmond.* W. L. Banks, Jr., PhD, Box 37, MCV/VCU Cancer Center, Richmond VA 23298, (804) 786-0771.

March 20-24
Regional Meeting, American College of Cardiology, *New Orleans.* W. D. Nelligan, MD, 9111 Old Georgetown Road, Bethesda MD 20814.

continued over

MEETINGS continued

March 21-25
Second Annual Review of Critical Care Medicine. (Johns Hopkins University), *Baltimore*. Cat. I credits. Fee: $450. Noreen Javornik, Office of CME, 720 Rutland Avenue, Baltimore MD 21205.

March 24-26
Gynecologic Oncology (Johns Hopkins University), *Baltimore*. Cat. I credits. Fee: MDs, $300; residents, $150. Susan Bavaro, Office of CME, 720 Rutland Avenue, Baltimore MD 21205

March 25-27
Audiovisual Symposium on Short Term Dynamic Psychotherapy (Virginia Institute of Short Term Dynamic Psychotherapy), *Charlottesville*. 21 Cat. I credits. Fee: $150. Jason Worchel, MD, 1932 Arlington Blvd., Charlottesville VA 22903, (804) 977-6644

March 27
Sports Medicine for Women (Eastern Virginia Medical School), *Norfolk*. Lou Ann Hamer, EVMS, PO Box 1980, Norfolk VA 23501.

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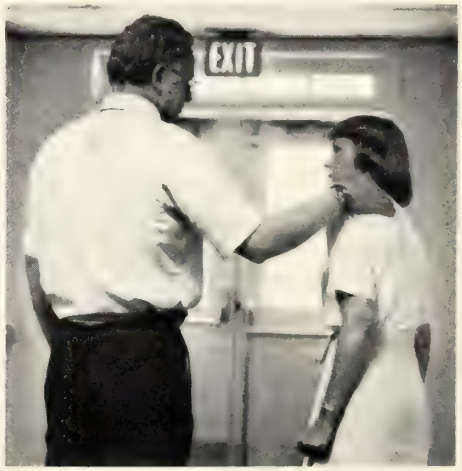

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1. National Interagency Council on Smoking and Health. *The Smoking Digest* Progress Report on a Nation Kicking the Habit. 1977

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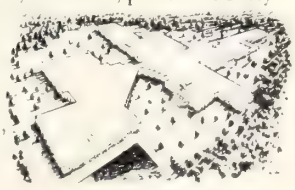

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VIRGINIA
MEDICAL
JANUARY 1983

The Case for "Tight Control" of Diabetes Mellitus

Robert M. Centor, MD, and David F. Gardner, MD, *Richmond, Virginia*

Patients enthusiastically endorse home glucose monitoring, the authors find, because they, rather than the physician, exercise control of their disease. They can adjust both dosage and timing of their insulin therapy to suit their lifestyles, rather than vice versa. As the technique gains acceptance, the authors suggest, a reduction in the incidence of complications may occur.

THE proper management of diabetes mellitus has been a subject of controversy among internists over the past two decades.[1,2]

One side in this controversy suggests that the long-term complications of diabetes are secondary to the metabolic derangements to which the patient is subjected over the course of the disease. They argue that correcting or improving the metabolic abnormality will diminish or postpone the number and severity of the complications.[3,4]

Others contend that such treatment has not been proven to prevent or ameliorate complications and that the potential side effects of aggressive insulin therapy may indeed be more harmful than the potential benefits.[5,6]

At present, there are no well-designed, randomized, prospective studies in humans to resolve definitively this issue, although reasonable conclusions can be drawn from a variety of animal and human investigations.[7-9] This discussion will delineate the controversy, present the case for "tight control," and discuss methods of achieving tight control in the 1980s.

From the Divisions of Primary Care and General Medicine and the Division of Endocrinology, Department of Internal Medicine, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Centor at Box 102, MCV Station, Richmond VA 23298.

This article was presented in slightly different form at Grand Rounds of the Department of Internal Medicine, Medical College of Virginia, on October 27, 1981.

The Controversy

There is general agreement that diabetic microangiopathy (retinopathy and nephropathy) and neuropathy increase in incidence, prevalence and severity as the duration of diabetes increases. Pirart, in his classic longitudinal study of 4,400 patients over 26 years, has amply demonstrated these relationships.[10] What is not agreed upon is the basis of this correlation. As with any correlation, there are two possible explanations. A change in one variable may lead to a change in the other variable (e.g.,

hyperglycemia causes complications), or there may be a third (or lurking) variable which is responsible for the changes in both variables. This latter explanation is consistent with the hypothesis that there is a direct genetic basis for diabetic microangiopathy, unrelated to the metabolic derangement.

This hypothesis is based partly on studies reported in 1968 by Siperstein and his coworkers, who measured the thickness of quadriceps muscle capillary basement membranes in diabetic patients, in normal controls, and in "prediabetics" (subjects with a known genetic predisposition to diabetes).[11] They found that almost all of the diabetic patients had thickened basement membranes, while less than 10% of the normal subjects had thickened basement membranes. Half of the "prediabetics" had thickened membranes. This work was interpreted as demonstrating that the microangiopathy of diabetes mellitus precedes the metabolic derangement. It was concluded that the microangiopathy is a hereditary abnormality associated with but not caused by the metabolic disturbance. It is this study that the opponents of tight control frequently cite as evidence that tight control will not necessarily prevent the complications of diabetes mellitus.

Proponents of tight control take issue with this study. Other investigators have been unable to reproduce Dr. Siperstein's findings, and the debate over methodological issues has continued for over ten years. Other investigators in this field have not found any increase in basement membrane thickness in prediabetics, and many experts now discount the importance of the Siperstein study. Most studies, in fact, show that capillary basement membrane thickening is related to the duration of diabetes, supporting a role for the metabolic abnormality in the development of this classic pathological feature of diabetic microangiopathy.[7-9]

The Case for Tight Control

The case for tight control can be made in two ways. First, we should demonstrate that the altered metabolic state associated with diabetes mellitus can lead to complications. Second, we should demonstrate that ameliorating this "dysmetabolism" can lead to a decrease in these complications. Several lines of evidence will be marshalled to further this argument. Animal studies are available which have addressed these questions in a controlled manner. Human studies involving monozygotic twins and patients with "acquired" diabetes (e.g., hemochromatosis, pancreatectomy, etc.) provide additional confirmatory data. Finally, the transplantation literature provides us with yet another opportunity to explore the relative contribution of genetic and metabolic factors in the pathogenesis of diabetic complications.

There are several methods for inducing diabetes mellitus in normal rats. Because of the availability of inbred strains of rats which are genetically identical, it is possible to perform randomized controlled studies in which the only significant variable is the artificially-induced diabetic state. These diabetic rats develop the typical pathological changes of diabetic renal disease, and it is reasonable to conclude that the glomerulopathy is secondary to the altered metabolic state, since genetic and environmental factors have otherwise been held constant.[12] Similarly, in dogs made diabetic either by injections of bovine growth hormone or alloxan, there is a consistent finding of increased capillary basement membrane width in kidney, retina and muscle.[13] Control dogs show no such changes. Because the microscopic lesions seen in these diabetic animals are not identical to those seen in humans, similar data in man are needed.

Ireland examined renal biopsies in ten patients with "acquired" diabetes and nine patients without diabetes.[14] While all of the nondiabetic patients had normal renal capillary basement membrane thickness, seven of the ten patients with acquired diabetes had thickened basement membranes. Raskin studied eight patients with pancreatic diabetes using the technique of Siperstein.[7] All patients were studied serially with a mean interval of five years. While originally only one patient had abnormally thickened basement membranes, five of the patients had abnormal thickness, and two others showed a significant increase in thickness at followup examination.

Studies with identical twins provide important information regarding the relative contributions of genetic factors and a disturbed metabolic environment to diabetic complications. In a study of ten pairs of identical twins discordant for diabetes, five of the twins with diabetes had retinopathy, while none of the twins with normal glucose metabolism was affected.[15] In another study, one twin had had diabetes for 16 years, while her sister had normal glaucose metabolism. The diabetic twin had markedly thickened muscle capillary basement membranes; her sister was entirely normal.[16]

Interesting results have also been found in transplantation studies. When a kidney is transplanted from a normal rat into a diabetic rat, the transplanted kidney develops a glomerulopathy.[17] This finding supports the concept that the diabetic milieu is responsible for the morphological changes, not he-

redity or the agent which originally induced the diabetes. This work has been reproduced in humans. Forty renal biopsies of successful kidney transplants were examined. Transplanted kidneys in ten of the 12 diabetic patients showed vascular changes of diabetes within five years of the time of transplantation, while only three of the 28 nondiabetic patients developed renal lesions consistent with diabetes (and only after five years).[18]

These studies clearly support the concept that the microangiopathic changes seen in diabetes are secondary to the disordered metabolism. We will now review the evidence that ameliorating the abnormal metabolic state prevents or postpones the complications of diabetes.

A significant body of evidence in favor of tight control comes from the transplant literature. When kidneys are taken from diabetic rats and transplanted into nondiabetic rats, there is significant improvement in the renal histologic lesion. While this occurs only if the transplantation is done early in the course of the diabetes, it does suggest that there is at least partial reversibility of the diabetic lesions. Similarly, if pancreatic islet cells are transplanted into diabetic rats early in the course of the renal disease, resulting in a "cure" of the diabetes, the renal lesions improve as well.[19]

In a followup of the previously mentioned dog studies, diabetic dogs were randomly assigned to tight control versus "loose control" groups.[20] One group of dogs was given insulin twice daily, resulting in "good control" (infrequent hyperglycemia and glycosuria). The other dogs received insulin once a day with resultant "poor control" (chronic hyperglycemia). The good control dogs developed significantly less retinopathy, nephropathy and capillary basement membrane thickening than the poor control dogs. If this model can be extrapolated to man, the concept of tight control to prevent the complications of diabetes is strongly supported.

Pirart, in his longitudinal studies, assessed the control of the patients yearly.[10] While not a randomized study, he did find significantly more end organ damage in the poorly controlled patients as compared with the more tightly controlled patients. In a controlled study, Job and coworkers randomized patients with diabetic retinopathy to once daily or multiple daily insulin injections.[21,22] Despite a less than perfect study design, they were able to demonstrate less of a yearly increase in the number of microaneurysms diagnosed by angioflurograms in the multiple injection group. Finally, Viberti et al have recently demonstrated that short-term, strict, metabolic control, even in long-standing diabetes, is associated with a diminution in urinary albumin excretion.[23]

While a detailed discussion of diabetic neuropathy is beyond the scope of this review, it should be noted that in both experimental animals and humans, abnormalities of nerve conduction velocity can be reversed by improvement of diabetic control. In addition, in at least one study of human diabetes, there was a significant positive correlation between the severity of the defect in nerve conduction and the degree of hyperglycemia.

Taken together, these studies strongly suggest that improving the metabolic environment of diabetic patients may decrease the incidence and severity of diabetic microangiopathy. (There is no such evidence regarding macroangiopathy.) Next we must examine the feasibility of achieving improved control in our patients.

Can We Achieve Tight Control?

First we must define tight control. The goal of therapy should be the maintenance of a blood glucose profile indistinguishable from individuals without diabetes mellitus.[24] While the goal is unrealistic at the present time, therapy in the 1980s should be directed toward approximating this ideal, at the same time avoiding intolerable side effects and disturbance of life style.

There are several methods for assessing glycemic control. The most commonly used is urine glucose monitoring. The principle underlying this approach is that a renal threshold exists for glucose, above which glucose will appear in the urine. The amount of glucose in the urine can be estimated by the patients using a variety of simple, semi-quantitative techniques. Although the average renal threshold is a serum glucose concentration of 160-180 mg/dl, there is considerable variation between patients and even from day to day in the same patient.[25] Additional drawbacks to this methodology are (1) a positive test correlates poorly with the degree of hyperglycemia, and (2) a negative test is difficult to interpret, as it may be associated with a blood glucose in the frankly hypoglycemic range or one just below the renal threshold.

Because of the inherent difficulties associated with urine glucose monitoring, outlined above, home glucose monitoring has recently become a popular method for monitoring diabetic control.[26] The technique involves placing a drop of capillary blood obtained by fingerstick onto test strips impregnated with glucose oxidase and an indicator dye. Strips can be read visually or by a reflectance meter. These strips are extremely accurate when

compared with standard laboratory plasma glucose or whole blood glucose methods.[27] The only disadvantages of these methods are the expense of supplies and the discomfort of the fingerstick. With the availability of automatic lancets, the discomfort of repeated fingersticks is minimal for most patients. The cost can be kept to approximately 35¢ per measurement. The obvious advantage is that an accurate record can be obtained of the patient's pattern of blood glucose response to a particular dose and type of insulin. The availability of multiple glucose measurements at home and at work enables the physician and patient to make rational adjustments in the insulin regimen and avoids overdependence on isolated determinations in the physician's office. The net result is greater patient involvement in the management of the disease and almost invariably improved diabetic control.

Many physicians have advocated the use of hemoglobin A_{1c} (glycosylated hemoglobin) measurements to assess control of diabetes.[28] Nonenzymatic glycosylation of hemoglobin occurs over time in proportion to circulating plasma glucose concentrations. A single hemoglobin A_{1c} determination provides a good estimate of "average" blood glucose levels over the preceding 6-8 weeks. Several studies have documented a good correlation between hemoglobin A_{1c} levels and 24-hour urinary glucose excretion or mean fasting blood glucose levels. This measurement may be useful in studying objectively the long-term effects of tight control on the development of microangiopathy.[29] However, the precise role of hemoglobin A_{1c} measurements in clinical practice remains to be defined.

Assessing control is one matter, but achieving tight control is another. The most common method of insulin administration is a once daily injection of an intermediate-acting insulin (NPH or lente). While this can be an acceptable method for avoiding severe hyperglycemia, the degree of control achieved in many patients is unacceptable to the adherents of tight control. Various multiple dose insulin regimens have become popular over the past several years. These have ranged in complexity from twice daily combinations of NPH and regular insulin to what has been referred to as "intensive conventional therapy"—the administration of a long-acting insulin (PZI or ultralente) with 3-4 preprandial doses of a short-acting insulin. Coupled with home glucose monitoring, the schedules are compatible with excellent 24-hour blood glucose control.[30,31]

The insulin pump has received considerable attention in both the medical and lay press. The pump is a portable device which allows for a continuous slow basal insulin infusion into the subcutaneous tissue, augmented by insulin boluses prior to meals. Results achieved with these pumps have generally been very good, but comparative studies show no significant difference between "intensive conventional" regimens and the continuous infusion systems. Closed loop devices (artificial pancreas) and islet cell transplantation are important avenues of current research; however, they do not appear to be of any immediate value to our patients.[24]

The degree of glycemic control obtained with either multiple dosing regimens or continuous insulin infusion pumps, while not achieving the glucose profiles seen in non-diabetic patients, is considered to be excellent by diabetologists.[32,33] This belief is strongly supported by marked decreases in hemoglobin A_{1c} levels in patients on these regimens as compared to traditional regimens. Moreover, this improvement is achieved without an increase in hypoglycemic episodes.

At this time, multiple dose insulin regimens are easier and less costly to introduce to patients than external pump devices. While some patients will read about the insulin pump and desire this technology, the majority of patients in whom tight control is attempted should probably start with multiple injections and home glucose monitoring. This approach has achieved excellent patient acceptance.

Summary

The case for tight control has been made. It is very convincing, and there is no case for loose control. In most patients, home glucose monitoring appears to be a practical method of monitoring diabetic control and has been enthusiastically endorsed by many patients. In combination with multiple daily insulin injections, the patient establishes control of the disease, as opposed to the physician controlling the patient. Patients are able to adjust their insulin (both dosage and time of administration) to their lifestyle, as opposed to having to adjust their lifestyle to the diabetes. While this approach may not prevent all the complications of diabetes, one would anticipate a reduction in the incidence of diabetic complications as these techniques gain widespread acceptance. While research continues in the quest for better management options, there is much that we can do for our patients now.

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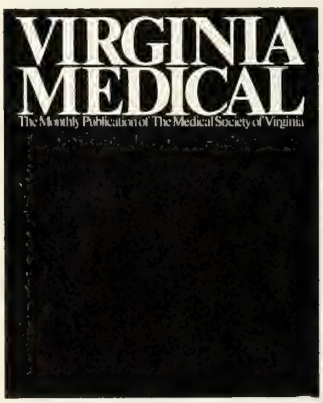

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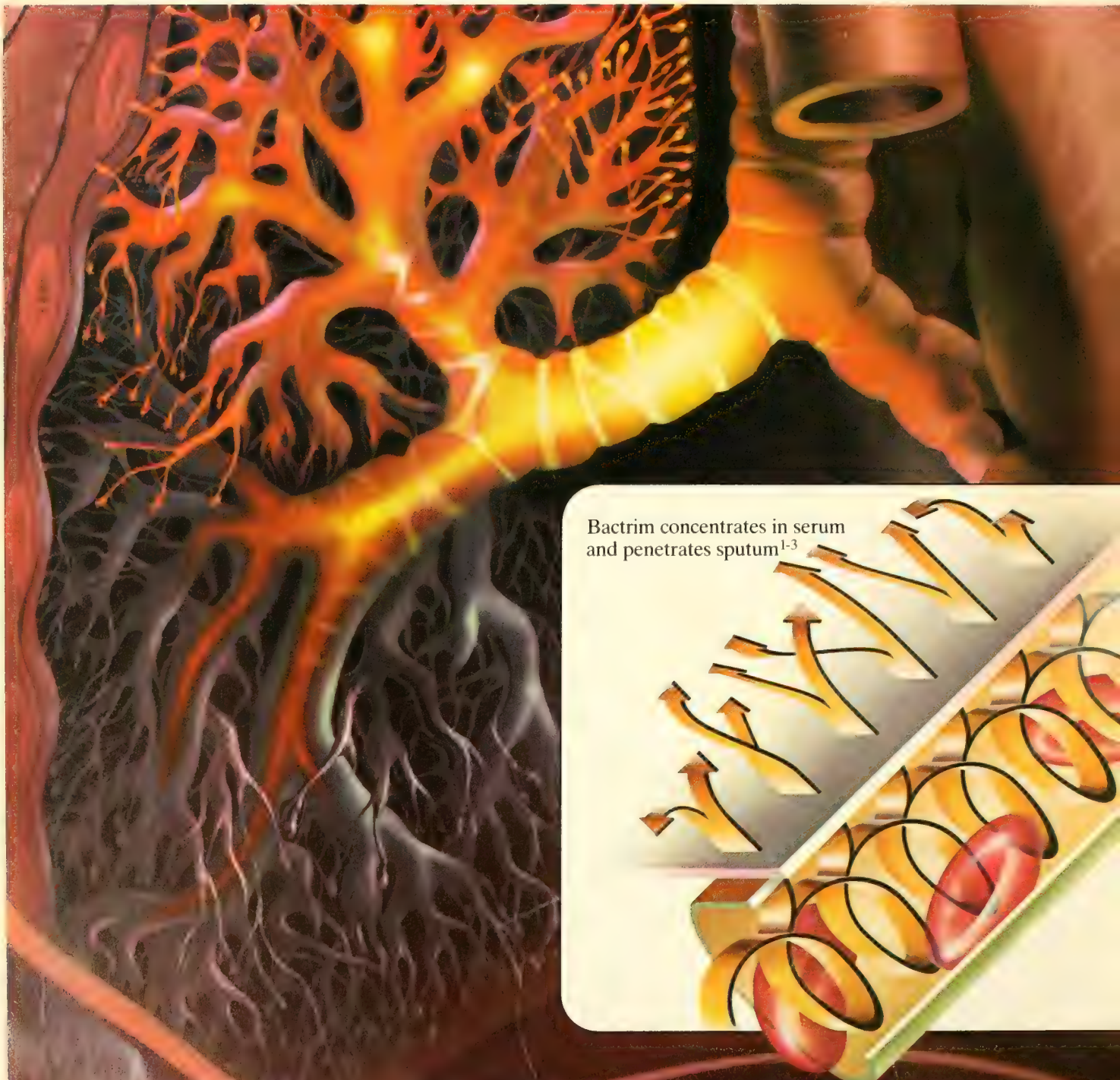

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ROCHE

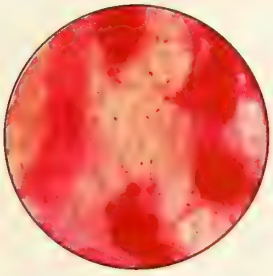
major pathogens of chronic bronchitis*

Bactrim clears sputum of susceptible bacteria

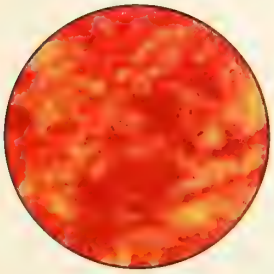
In sputum cultures from patients with acute exacerbations of chronic bronchitis, *H. influenzae* and *S. pneumoniae* are isolated more often than any other pathogens.[4,5] One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens.[5]

Bactrim is effective *in vitro* against most strains of both *S. pneumoniae* and *H. influenzae*—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.[6]

attacks *H. influenzae*—even ampicillin-resistant strains



attacks *S. pneumoniae*



Bactrim reduces coughing and sputum production

In three double-blind comparisons with ampicillin *q.i.d.*, Bactrim DS proved equally effective on all clinical parameters.[7-9] Bactrim reduced the frequency and severity of coughing, reduced the amount of sputum produced and cleared the sputum of purulence.

Bactrim has the added advantages of *b.i.d.* dosage convenience and a lower incidence of diarrhea than with ampicillin, and it is useful in patients allergic to penicillins.

Bactrim also proved more effective than tetracyclines in 10 clinical trials involving nearly 700 patients.[10] Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

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Economical b.i.d.

Bactrim™ DS

(160 mg trimethoprim and 800 mg sulfamethoxazole/Roche)

*Due to susceptible organisms. **Please see next page for summary of product information.**

Bactrim™

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note.* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects. Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions.* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions.* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions.* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment. Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS

Usual adult dosage. 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100, Tel-E-Dose® packages of 100; Prescription Paks of 20 and 28. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500, Tel-E-Dose® packages of 100, Prescription Paks of 40. *Pediatric Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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For Control of Chronic Pain, Electrical Stimulation of the Brain

Richard P. Greenberg, MD, and Donald P. Becker, MD,
Richmond, Virginia

The unraveling of complex mechanisms of pain inhibition in the central nervous system suggested a new surgical therapy for control of chronic pain: electrical stimulation of the periventricular region of the brain, where pain inhibition by both narcotic drugs and electrical stimulation is mediated.

PAIN caused by medical or surgical disease can generally be managed with appropriate treatment and judicious use of analgesic medication so long as it resolves within weeks to a few months of onset. However, if the painful process is not curable or self-limiting and persists for six months or more, a chronic pain syndrome can develop.[1,2]

Chronic pain syndromes are difficult to treat successfully and are often refractory to standard therapy.[1,2] We evaluated the efficacy of a new surgical procedure, electrical stimulation of a pain inhibitory center of the brain, the periventricular grey matter, in patients with a variety of chronic pain problems. This approach was suggested to us and others by aspects of the gate control theory of pain and recent advances in our understanding of the brain's descending inhibitory systems.[3-7]

The gate control theory of pain proposed in 1965 by Melzack and Wall attempted to summarize what was known at that time about pain inhibitory and facilitatory mechanisms within the central nervous system.[6] Three central systems that could modulate pain perception were proposed: 1) the spinal cord dorsal horn sensory transmission neurons (T-cells) that received input from both low and high threshold primary afferents; 2) substantia gelatinosa, spinal cord interneurons which modulated the excitatory affects from these primary afferents; and 3) *descending control systems* which originated within the brain.

Considerable progress has been made recently in describing the anatomical, physiological and neurohumoral substrates of these descending pain control systems.[4,5,8] It was noted that electrical stimulation of the periventricular grey matter produced profound analgesia in experimental animals.[4,9] Furthermore,

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the pain inhibitory effects of both narcotic drugs and brain electrical stimulation appear to be mediated by activity at the same brain site.[3] Both narcotic-produced analgesia and brain stimulation-produced analgesia are *reduced* by depletion of monoamines, e.g., serotonin, and by the narcotic antagonist naloxone. These two modes of pain suppression exert their effects by activation of the periventricular grey matter, which in turn stimulates a serotonergic pathway in the brain stem that descends via the dorsolateral funiculus of the spinal cord to the dorsal horn.[4,8] The ultimate site of inhibitory action involves the release of serotonin and its consequent inhibition of spinal cord dorsal horn (T-cell) neurons that are the origin of nociceptive pathways.

Methods

Patients presenting with intractable chronic pain must be meticulously screened by a multidisciplinary group to identify those cases best treated by further diagnostic evaluation or psychiatric consultation. The remaining chronic pain patients are often given a narcotic medication to determine if their pain levels fall with opiates because the pain inhibitory effects of narcotics and brain stimulation are mediated by activity of the same brain sites. An active placebo, e.g., nicotinic acid/NaCl, should also be utilized as a control drug to assure unbiased results. (Some chronic pain syndromes, such as phantom limb pain and anesthesia dolorosa, do not respond well to narcotic medications or electrical stimulation of the periventricular grey). In a group of 62 patients thought by us on clinical grounds to be good candidates for electrode implantation, only 21 were considered for surgery after psychiatric evaluation and drug test therapy.

Implantation of the electrode is performed in the operating room with the patient awake, having received only mild diazepam (Valium®) sedation. The head is placed in a stereotactic frame, a burr hole made, and contrast material, air or metrizamide is introduced into the third ventricle via a cannula. After locating the posterior commissure, a target is calculated that lies 2-3 mm lateral to the third ventricle at the level of the posterior commissure in the horizontal and anteroposterior planes (Fig. 1). A platinum-irridium electrode is introduced to the target in the brain through the burr hole. The distal end of the stimulating wire is tunneled subcutaneously for several inches under the scalp, then externalized.

For the next 3-5 days, the patient can self-stimulate via the externalized electrode wires and

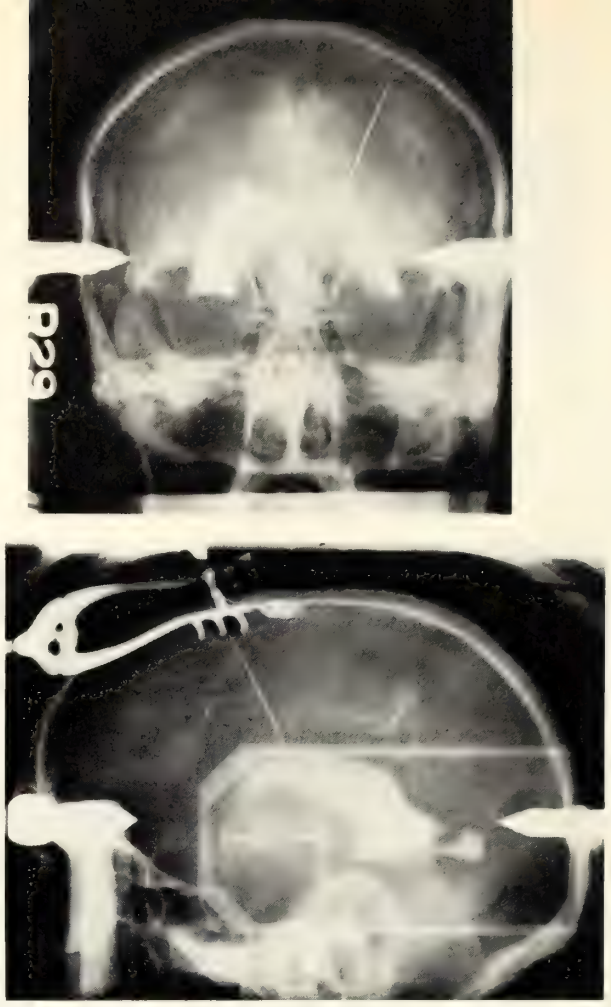

Fig. 1: Intraoperative X-rays during stereotactic implantation of periventricular grey electrode. Final placement of depth electrode is demonstrated in AP (top) and lateral (bottom) views. Contrast material still visible in ventricular system. Target localization is based on intraoperative ventriculogram. Electrode is 2-3 mm lateral to third ventricle at level of posterior commissure in AP and horizontal planes.

determine if the procedure has reduced the pain level. Permanent internalization of the unit is done only if periventricular stimulation has helped the patient over the 3-5 day test period. Thereafter, the internalized subcutaneous receiving portion, which is similar to a cardiac pacemaker unit, can be transcutaneously activated by a battery-powered external apparatus. Patients need stimulation for only 1-2 hours daily, as the heightened activity of the pain-suppressing periventricular brain site lasts for 23-24 hours.

Results

Of 21 patients who had stereotactic implantation of periventricular grey electrodes, 12 had chronic low back pain with multiple previous lumbar surgical procedures with and without proven arachnoidi-

tis, three patients had cervical and upper extremity pain with previous surgery of the cervical spine, two had atypical facial pain, one cervical cord astrocytoma, one cervical cord arachnoiditis, and one brain stem infarction with pain.

Reduction of clinical pain was most successful in the multiple-surgery, chronic low back pain group; 60% (7/12) overall were able to return to work and lead a drug-free life. Two additional patients in this group are still handicapped by their pain and have not returned to work but are drug-free. Only one of the three patients with cervical and upper extremity pain has had a good result from electrical stimulation. The patient with a cervical cord astrocytoma and one with cervical cord arachnoiditis were both well pleased with the level of pain reduction produced by brain stimulation. All three patients with atypical facial pain failed to get pain relief with this procedure. The patient whose pain syndrome followed a brain stem infarct considered the procedure a success, although he had periods of pain relief even when not stimulating.

In this series two patients developed intracerebral hematoma, verified by CT scan, that was clinically detected because of hemiparesis in one patient and an up-gaze palsy of the visual system in another. Both of these complications were transient and disappeared within seven days of the procedure. Complications reported by other groups performing this procedure include scalp infection, meningitis and death secondary to postoperative intracranial hematoma. The overall morbidity is less than 10% and the mortality is 1-2% for this procedure.

Discussion

Our group as well as others has noted effective reduction in chronic pain levels following electrical stimulation of the periventricular grey brain region.[3,7] This success would appear to be based on the augmentation of activity in a descending neural system originating in the brain that can inhibit pain. The same brain site is activated by narcotic medication and rendered ineffective by the narcotic antagonist naloxone.

However, stimulation-produced analgesia is not uniformly efficacious for all chronic pain syndromes. Our data as well as that of Hosobuchi et al[3] and Richardson et al[7] suggest that facial pain and certain cervical pain problems do not respond well to this type of surgical treatment. On the other hand, low back pain patients, in our experience and that of others, derive a great deal of pain relief from brain stimulation. Recent work in animal models suggests that there is more than one system of descending pain control and that a difference may exist between the upper extremities and lower extremities with regard to the respective descending system at work suppressing pain.[5,9] Thus, regional differentiation of descending pain inhibition uncovered in animal models may ultimately provide the answer to our findings in patients.

Electrical stimulation for pain control has advantages over lesions made in the peripheral or central nervous system. Stimulation therapy is reversible, whereas a CNS lesion is not. Moreover, unpleasant side effects, such as anesthesia dolorosa, that sometimes develop following CNS lesions can be avoided with stimulation therapy. However, analgesia produced by brain electrical stimulation is not the surgical procedure of choice for all patients with chronic pain. Patient selection is critical, as one must take into account psychological factors as well as the cause and location of pain.

Our findings suggest that chronic low back pain responds best to this procedure. As mechanisms governing descending pain control are further understood, stimulation-produced analgesia may have a wider application and become increasingly helpful in relieving chronic pain.

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Congenital Elliptocytosis with Red-Cell Cholinesterase Defect: Case Report

Mario Stefanini, MD,
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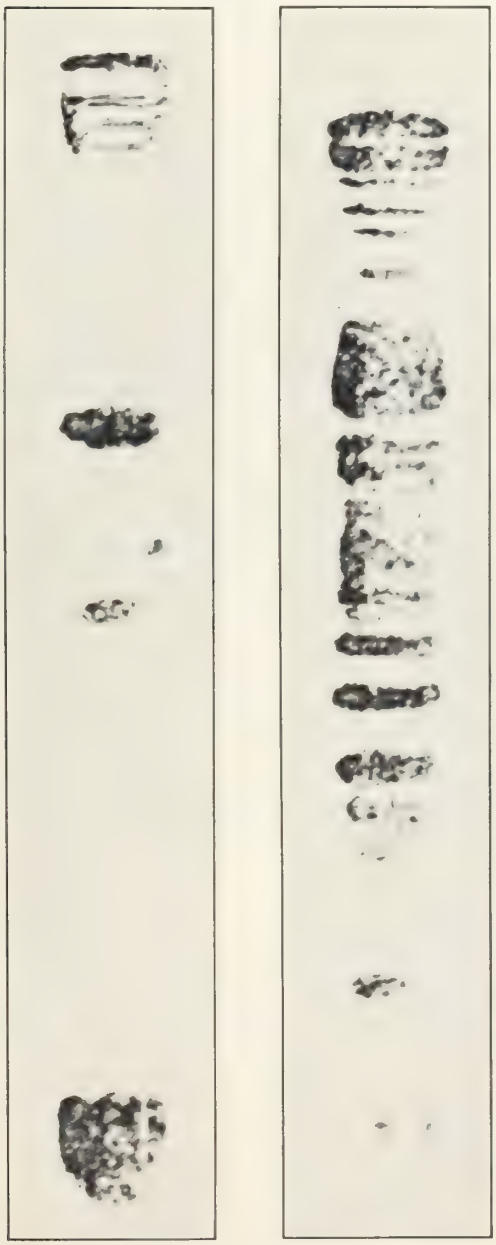
Richlands, Virginia



CONGENITAL elliptocytosis (ovalocytosis) is an anomaly transmitted as an autosomal dominant. It is characterized by the predominance in the peripheral blood of cigar-shaped red cells whose abnormal contours are obscure in origin. This clinical note describes the finding of an enzyme abnormality in the red cells of both a young adult with such a disorder and her offspring.

Case Report

A 15-year-old female was admitted because of intermittent and recurrent right lower quadrant abdomal pain of several years' duration, unrelated to menstrual periods. The past history included episodes of tonsillitis, earaches, impetigo and back pain attributed to flaring of the left iliac crest. For the previous four months the patient had suffered from blackout spells with visual central scotoma, attributed to a migraine equivalent since the symptoms were improved by the use of propranonol (Inderal®).

Physical examination showed a young woman in no severe distress. Blood pressure was 115/70 mm Hg, pulse 79/minute and regular. Eye grounds appeared normal. Heart and lungs were normal at percussion and auscultation. Lymph nodes, liver and spleen were not palpable. Examination of the abdomen and vagina was unremarkable. Neurological findings were within normal limits. An average blood count showed RBCs 4.51 M/cu mm; hemoglobin 13.7 gm/dl; hematocrit 39%; WBCs 10,900/cu mm with 57% neutrophils, 31% mature lymphocytes and 2% monocytes; platelets 501,000/cu mm; reticulocytes 1.8%. The peripheral blood smear, however, averaged 75% oval cells, 9% cigar-shaped cells, 1% spherocytes and 15% biconcave cells. Chemical parameters, parameters of thyroid function and tests of hepatic function by standard automated procedures were within limits of normal. A CAT brain scan was negative. Electroencephalo-

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Fig. 1. One-dimensional electrophoretic patterns of erythrocyte membrane proteins from patient in case report, using dodecylsulfate-polyacrylamide gel loaded with 50 μgs protein. Far left, Schiff's periodic acid stain. Near left, Coomassie brilliant blue stain. Same study using protein from normal person yielded essentially the same pattern.

Table 1. Results of Studies of Patients with Congenital Elliptocytosis.

	Patient	Average in 5 healthy controls
Metabolic Tests		
Osmotic fragility of red cells (% NaCl)		
initial	0.38	0.38
final	0.30	0.28
Mechanical fragility (% hemolyzed cells)	1.8	1.4
Heinz bodies test (% erythrocytes containing five or more bodies)	10.2	9.7
Glutathione stability test (GSH level, mgs/ml erythrocytes)	36.2	35.7
Orthocresol red screening test	negative	negative
Autohemolysis Test		
	Mean % autohemolysis after 48 hrs incubation at 37°C	
Compound added and final concentration:	Patient	Healthy Control
None	2.84	2.90
Glucose, 0.026 M	0.81	0.72
Adenosine triphosphate, 0.02M	1.08	1.02

Table 2. Phosphate Compounds of Fresh and Incubated Red Cells from Patient with Congenital Elliptocytosis (μMoles/100 ml erythrocytes).

Hours of incubation	AMP	ADP	ATP	2,3 DPG	HDP	MGP	WAP	Total P	IP
0	1.3	19.8	143	358	24	2.1	42	1274	24
3	4.0	13.7	127	361	23	2.3	46	1291	49
6	3.2	13.6	114	204	24	2.0	43	1188	201
9	19.7	18.6	51	106	23	2.0	40	1203	603
12	42.1	16.0	46	86	18	1.9	38	947	587

AMP = adenosine monophosphate; ADP = adenosine diphosphate; ATP = adenosine triphosphate; 2,3, DPG = 2,3 diphosphoglycerate; HDP = hexose diphosphates; MGP = monophosphoglycerate; WAP = weak acid phosphate; IP = inorganic phosphate.

Table 3. Activity of Various Enzymes in Patient's Erythrocytes.

	Patient	Average and range in 5 healthy persons
Hexokinase*	0.23	0.21 ± 0.07
Phosphoglucose isomerase*	12.6	11.9 ± 2.1
Phosphofructose kinase*	3.76	4.01 ± 0.9
Aldolase*	1.02	0.78 ± 0.12
Triose-P-isomerase*	257.	234.6 ± 13.9
Glucose-6-phosphate dehydrogenase*	3.0	2.0 ± 0.5
Glyceraldehyde 3-P dehydrogenase*	14.2	15.6 ± 2.8
Monophosphoglycerate kinase*	27.2	29.0 ± 4.8
3-phosphoglycerate mutase*	10.8	11.3 ± 2.9
2,3 diphosphoglycerate mutase*	5.8	6.3 ± 1.1
Pyruvate kinase*	2.72	2.95 ± 0.6
Lactic dehydrogenase*	28.4	29.4 ± 3.7
Glutathione reductase*	1.4	1.5 ± 0.17
Glutathione peroxidase*	1.86	1.89 ± 0.09
Glutathione synthetase*	3.4	3.6 ± 0.6
Adenosine triophosphatase*	2.08	2.07 ± 0.3
Methemoglobin reductase*	0.36	0.39 ± 0.02
6-Phosphogluconate dehydrogenase*	1.39	1.33 ± 0.03
Enolase*	4.6	4.7 ± 1.0
Glyoxalase I**	49.7	51.0 ± 3.2
Glyoxalase II***	18.6	17.0 ± 2.1
Cholinesterase ****	4.7	10.1 ± 0.9

* results expressed as millimoles of substrate utilized/minute/10^{10} erythrocytes
** results expressed as μMoles of thioester formed/minute/10^{10} erythrocytes
*** results expressed as enzyme units (1 unit = disappearance of 1 μMole of thioester/minute/10^{10} erythrocytes)
**** results expressed as units (μMoles/minute/ml erythrocytes)

gram showed grade III dysrhythmia with left frontal spike, predominantly during sleep. No satisfactory explanation was found for the symptom of pain, which gradually subsided.

Because of the unexpected finding of marked ovalocytosis, additional studies of the chemical structure and metabolism of the patient's red cells were carried out.

Materials and Methods

Blood counts were obtained with a Coulter Counter S Plus. Thin and well-spread smears were stained with Wright's. Red cell osmotic[1] and mechanical[2] fragility, Heinz bodies test, glutathione stability test and orthocresol red screening test were performed as described previously[3]. Rate of autohemolysis with and without addition of glucose and of adenosinetriphosphate (ATP)[4]; levels of phosphate compounds, including adenosinemonophosphate (AMP), adenosinediphosphate (ADP), ATP, 2, 3 diphosphoglycerate, hexose diphosphates, monophosphoglycerate, weak acid phosphates and inorganic phosphates were also analyzed[5]. Levels of hexokinase, phosphoglucose isomerase, phosphofructose kinase, aldolase, triose-P-isomerase, glucose-6-phosphate dehydrogenase, glyceraldehyde 3-P-dehydrogenase, monophosphoglycerate kinase, 3-phosphoglycerate mutase, pyruvate kinase, lactic dehydrogenase (LDH), glutathione reductase, peroxidase and synthetase, adenosine triphosphatase, methemoglobin reductase, 6-phosphogluconate dehydrogenase and enolase were determined in red cell stroma using the techniques previously described[3]. Also determined in the red cells were 2-3 diphosphoglycerate mutase, glyoxalase I and II[6] and cholinesterase[7]. Protein and glycoprotein electrophoretic pattern of the constituents of the red cell membrane were also studied using "ghosts" (preparations of erythrocyte membrane), obtained as described by Steck et al[8]. One-dimensional electrophoresis with dodecylsulfate polyacrylamide gel[9] was used.

Results

Mechanical and osmotic fragility, Heinz body test, glutatione stability, orthocresol red screening tests and autohemolysis test (Table 1) were within limits of normal. Levels of phosphate compounds before and during incubation (Table 2) were comparable to those of red cells from normal individuals. All enzymatic constituents of red cells studied (Table 3) and the chromatographic pattern of proteins and glycoproteins of the red cell membrane (Figure 1) were within the ranges of values obtained in five apparently healthy individuals who did not show elliptocytes in their blood.

The only exception to normal was the cholinesterase level. A survey among 25 apparently healthy individuals in this area gave a mean normal titer of 10.1 μM/minute/ml erythrocytes with a range of 9.2 to 11.0. Five weekly determinations in the patient gave an average value of 4.7 μM/minute/ml erythrocytes with a range of 3.9 to 5.1.

The peripheral blood smears of the patient's mother and maternal half-brother showed no elliptocytosis. The father was not available for study.

About one year after these studies were made, the patient gave birth to a daughter, her first child. At birth, 69% of the red cells seen in the peripheral blood of the child were elliptocytes, and there were 12% nucleated red cells. The level of red cell cholinesterase titer was 1.5 μM/minute/ml. There was no evidence of acute or chronic hemolytic process. These findings establish both defects as congenital and, in all likelihood, hereditary.

Discussion

Irrespective of the number of abnormally shaped cells found in their peripheral blood, very few patients with congenital elliptocytosis show anemia and hemolysis; a few may show compensated hemolysis, but the majority give no indication of anemia or hemolysis, nor do they give evidence of biochemical or enzymatic defects of the erythrocytes. Although a membrane defect has been postulated to exist in elliptocytosis[10], the membrane protein pattern has been found to be normal.

The finding of red-cell cholinesterase deficiency in our patient may therefore be unique, and it gives rise to a question of the relationship of the enzyme defect to the morphological anomaly of the patient's red cells. The deficiency of another enzyme, glyoxalase II, has been found in cases of congenital elliptocytosis and thought to be coincidental.[6] Similarly, we have no ground to postulate a direct relationship between the deficiency of cholinesterase and the abnormal shape of the erythrocytes in our patient. Until other instances of such an association are reported, this finding in our patient may be considered to be coincidental and a result of multiple gene-related abnormalities.

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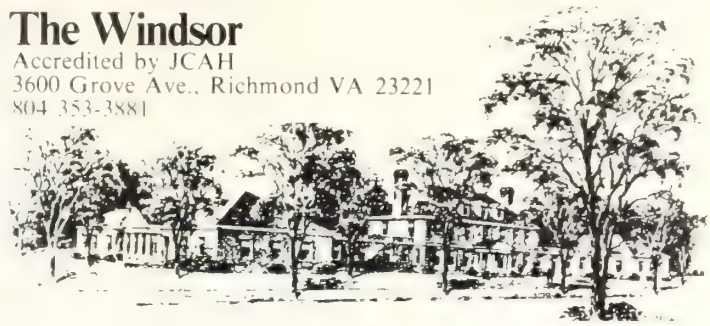

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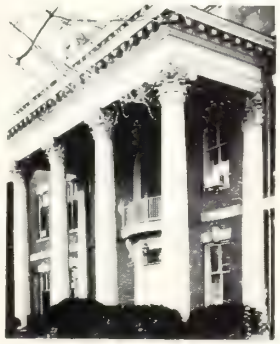

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Fusion of Anterior Cervical Spine Without Rigid Strut: Case Report

Richard Whitehill, MD,
Meredith Thomasson, MEd, and
Gwo-Jaw Wang, MD,
Charlottesville, Virginia

THE orthopedic literature contains numerous descriptions of anterior reconstruction techniques following excision of a portion of a cervical vertebral body to decompress the traumatized cervical spinal cord.[1-3]

One underlying assumption is that omission of the insertion of a rigid strut to hold the cavity left open by the vertebral body excision (the decompression defect) could result in unacceptable kyphosis and collapse, possibly ultimate instability and further spinal cord damage. The case reported herein raises some questions about this assumption.

Case Report

R. H., a 48-year-old black male, was admitted to the University of Virginia Hospital after sustaining quadriplegia in a motor vehicle accident. His admitting X-rays showed a flexion-axial loading type of fracture of the C5 vertebral body. In addition, a fracture into the posterior spinous process of C4 was also noted. (Fig. 1A).

The X-rays and a subsequent myelogram were interpreted as showing spinal cord compression secondary to retropulsion of the posterior aspect of the C5 body. It was felt that it would be possible to improve surgically the patient's neurological level of function by one nerve root. He therefore underwent an anterior C5 decompression and reconstruction with a full thickness iliac crest graft approximately ten days following his initial injury. Immediately after the operation the patient was placed in a halo vest (Fig. 1B).

The patient did well until the 6th postoperative day, when he complained of neck pain. An X-ray at this time showed that the iliac crest graft had migrated anteriorly. Because the patient also noted dysphagia, a Barium swallow was obtained, which showed contrast material outside of the esophagus at the level of the protruding iliac crest graft (Fig. 1C). The graft was excised and the wound drained. The patient was maintained in a halo vest. Luckily his wound healed without difficulty.

An X-ray obtained three months following graft excision and at the time of removal of the halo vest demonstrated that the decompression defect had been spontaneously bridged with bone (Fig. 1D). Lateral flexion and extension X-rays confirmed that the spine was stable. The patient had no further neck pain and his X-rays did not change in the succeeding 18 months. Neurologically, however, his last functioning myotome was unchanged from its preoperative status.

Discussion

The purpose of inserting the anterior bony strut in the decompression defect is twofold. It is positioned as a physical strut to resist collapse and kyphotic angulation of the adjacent vertebral bodies. It is also a bone graft, and as such it exists to become a fusion mass welded solidly to the adjacent vertebral bodies to provide ultimate spinal stability.

This case report indicates that in some cases it is possible to achieve both purposes without the presence of the physical strut. It was replaced by a halo vest which served orthotically as the strut would have internally. However, it should be noted that the transient presence of the graft and inflammation secondary to the esophageal injury probably served as osteogenic stimuli, which ultimately resulted in a solid fusion. Thus, broad generalizations regarding anterior cervical spine fusions should not be made from this one case report.

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Submitted 1-12-82.

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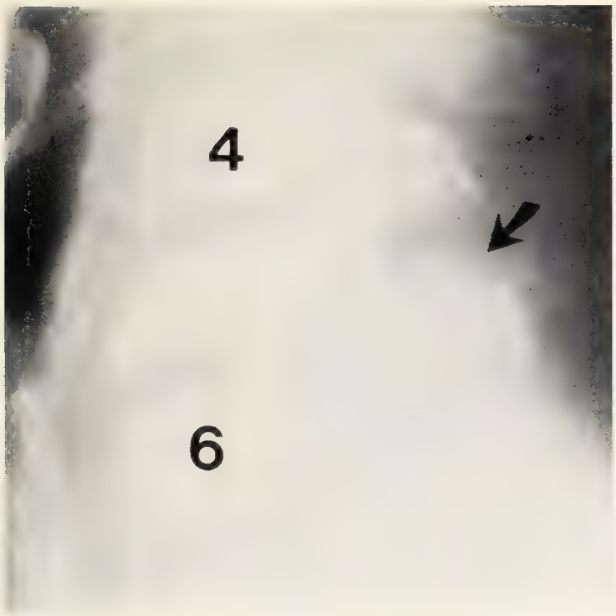

Fig. 1A. Lateral cervical spine X-ray showing flexion-axial loading type C5 injury. Mild bony retropulsion of C5 into spinal canal. Arrow points to fracture through base of C4 posterior spinous process.

Fig. 1B. Lateral cervical spine X-ray obtained in recovery room immediately following anterior C5 decompression and insertion of full thickness iliac crest bone graft.

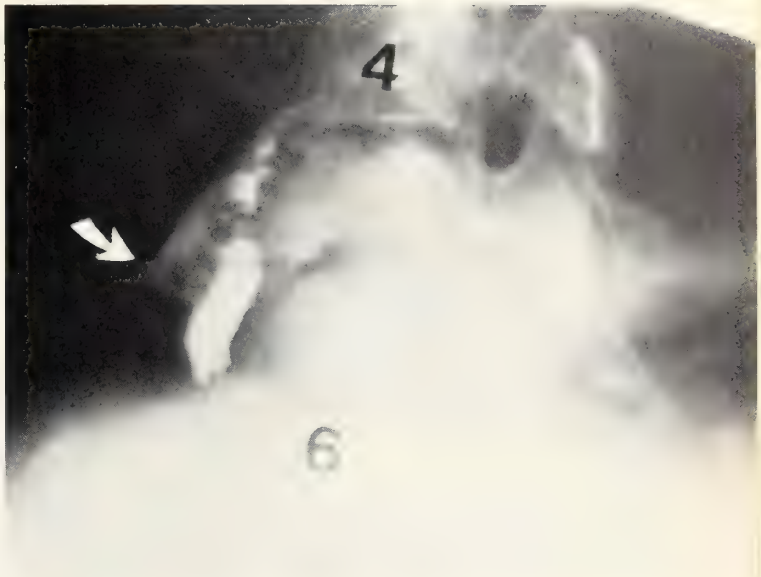

Fig. 1C. Lateral cervical spine X-ray obtained after barium swallow, showing contrast material outside esophagus adjacent to iliac crest graft. Arrow points to iliac crest graft, which has migrated anteriorly.

Fig. 1D. Lateral cervical spine X-ray obtained some three months following graft excision shows bony bridge spanning decompression defect from C4 to C6.

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Emergency Treatment of Hypoglycemia in Children

Robert D. Lovinger, MD,
Richmond, Virginia

CLINICALLY, hypoglycemia is considered present when two or more sequential *whole blood* glucose levels fall below 30 mg/dl in the normal newborn; 20 mg/dl in the premature infant during the first 24 hours of life; and 40 mg/dl in the normal child and adolescent. (Serum values are 10-15% higher than blood values).[1,2]

Carbohydrate Homeostasis

Blood glucose is maintained within normal limits by the interaction of 1) adequate supplies of glucose and gluconeogenic substances such as amino acids, lactate, and glycerol, provided through diet and mobilization from tissue stores; 2) functionally intact glycogenolytic and gluconeogenic enzyme systems in the liver; and 3) an efficient endocrine system for integrating and modulating glycogenolysis and gluconeogenesis.[2]

Fasting adults are able to maintain normal blood glucose levels for weeks. Infants and children, however, become hypoglycemic after even brief fasts because their glycogen stores are sufficient to meet obligatory glucose requirements for only 8-12 hours, possibly because of the disproportionate brain requirement relative to liver storage capacity in this age group. Once glycogen stores are depleted, glucose is derived primarily by gluconeogenesis of amino acids and glycerol. However, muscle mass and fat stores, the major source of amino acids and glycerol, are limited in children compared to those of the adult.

Hormones regulate glucose homeostasis. Insulin lowers the blood glucose by facilitating glucose entry (along with potassium) into cells. Four other hormones, on the other hand, elevate the blood glucose by a variety of mechanisms; epinephrine induces release of liver glucose by increasing the rate of glycogenolysis and directly suppresses insulin secretion as well; glucagon enhances both gluconeogenesis and glycogenolysis; cortisol and growth hormone not only increase glycogenolysis but also inhibit peripheral glucose utilization and elevate glycerol levels by stimulating lipolysis. In summary, hormone interaction maximizes available glucose for the brain.

From the Departments of Pediatrics and Pathology, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Lovinger at 6716 Patterson Avenue, Richmond VA 23226.

Submitted 8-19-82.

Neonatal Hypoglycemia

In the newborn period, hypoglycemia can generally be categorized as either transient or permanent. Transient hypoglycemia usually occurs within the first 48 hours of life. Infants who are premature, small for gestational age or under stress may develop hypoglycemia as a result of depleted glycogen stores and increased brain/liver ratio.[3,4] Infants of diabetic mothers and neonates with erythroblastosis fetalis, on the other hand, become hypoglycemic from hyperinsulinism. In infants of diabetic mothers, the hyperinsulinism is secondary to increased placental glucose transport.[5] Although glucose transport ceases at birth, the elevated serum insulin levels persist for one to two days. The cause of hyperinsulinism in erythroblastosis fetalis is unknown.

Permanent neonatal hypoglycemia may result from severe hereditary liver enzyme deficiencies, a form usually characterized by growth retardation and hepatomegaly.[6] Alternatively, it may arise from hyperinsulinism secondary to beta cell hyperplasia, tumor, or proliferation of beta cells along with pancreatic ducts, known as nesidioblastosis.[7] Hypoglycemia also occurs in Beckwith-Wiedemann syndrome (large infant with omphalocele, macroglossia, hepatomegaly, renal enlargement).[8]

Neonatal hypoglycemia is usually signaled by tachypnea, tremors, apnea, cyanosis, convulsions, hypothermia, or sudden unresponsiveness. At least two sequential blood glucose determinations in the hypoglycemic range are necessary to confirm the diagnosis. Although many schemes are available for the treatment of hypoglycemia, that of Cornblath is preferred (see box).[1] Unresponsive patients may need glucocorticoids in stress doses. Subsequent treatment depends upon the underlying disorder. For insulin hypersecretion syndromes, diazoxide and/or epinephrine may be useful, whereas growth hormone may be effective in patients refractory to these drugs.[1] For the infant of a diabetic mother, glucagon may alleviate the hypoglycemia until the infant attains stable, normal insulin levels. Gluca-

gon may be administered intravenously or intramuscularly in a dose of 300 mg/kg IV or IM, not to exceed 1 mg.[1] If the patient fails to respond, the dose should be repeated in four minutes.

Hypoglycemia in Childhood

The commonest symptomatic hypoglycemia in childhood is ketotic hypoglycemia.[9] It is seen most often in thin toddlers with a history of low birth weight for gestational age in whom food deprivation or infection characteristically precipitates hypoglycemia, accompanied by ketosis, vomiting and seizures. Although the etiology of ketotic hypoglycemia is not specifically known, it appears to be the result of limited liver glycogen stores and an inability to mobilize gluconeogenic precursors adequately. Lack of patient response to glucagon during a hypoglycemic episode, but not otherwise, distinguishes this condition. Treatment involves frequent feedings during the day, and bedtime snacks. Less common causes include deficiencies of growth hormone or of cortisol,[11] hepatic enzyme deficiencies,[10] hepatitis,[12] Reye syndrome,[13] insulin-secreting pancreatic tumors, Wilms tumor or neoroblastoma, and ingestion of aspirin,[14] alcohol[15] or other hypoglycemia-potentiating drugs.

Therapy for the toddler and older child is specified in the accompanying box, including stress dose if response is not immediate.

Continued therapeutic failure may indicate the presence of hypoglycemia-induced cerebral edema, a diagnosis that must also be considered in any patient who becomes biochemically normal after treatment for diabetic ketoacidosis yet whose mental status fails to regain a pre-crisis mental status.

TREATMENT OF ACUTE HYPOGLYCEMIA

Neonate

Initial: Rapidly infuse IV glucose (25% solution in water) 1-2 ml/kg. Continue infusion at 6-8 mg/kg/min using 10-15% glucose solution. Adjust infusion rate to maintain blood glucose values 50-100 mg/dl, monitoring with reagent strips (Dextrostix® or Chemstrip bG®) at least each hour. After blood glucose levels stabilize at 6-12 hrs, glucose rate and concentration may be reduced over 6-hr period while feedings are resumed.

Comments: 1) Administer glucose concentrations more than 10% through central line. 2) Monitor electrolytes and hydration carefully. Obtain pediatric consultation if imbalance occurs.

Refractory (stress dose): Increase glucose infusion rate to 12-24 mg/kg/min. If necessary, use Solu-Cortef®-45 mg/M^2 initially and 100 mg/M^2/day in infusion bottle for continuous treatment.

Comments: 1) May need central line. 2) Wean patient as soon as possible, but slowly since abrupt discontinuation may precipitate fatal reactive hypoglycemia.

Child

Initial: Rapid IV delivery of 0.5 gm/kg of 50% glucose diluted with equal volume of water (D25), not to exceed 25 gm. Maintain child on 10% glucose in 0.25% normal saline to keep blood glucose greater than 40 mg/dl.

Comments: 1) May need central line. 2) Monitor electrolytes and hydration carefully. Obtain pediatric consultation if imbalance occurs.

Refractory: Use glucocorticoids as suggested for neonate.

Comment: Watch for cerebral edema.

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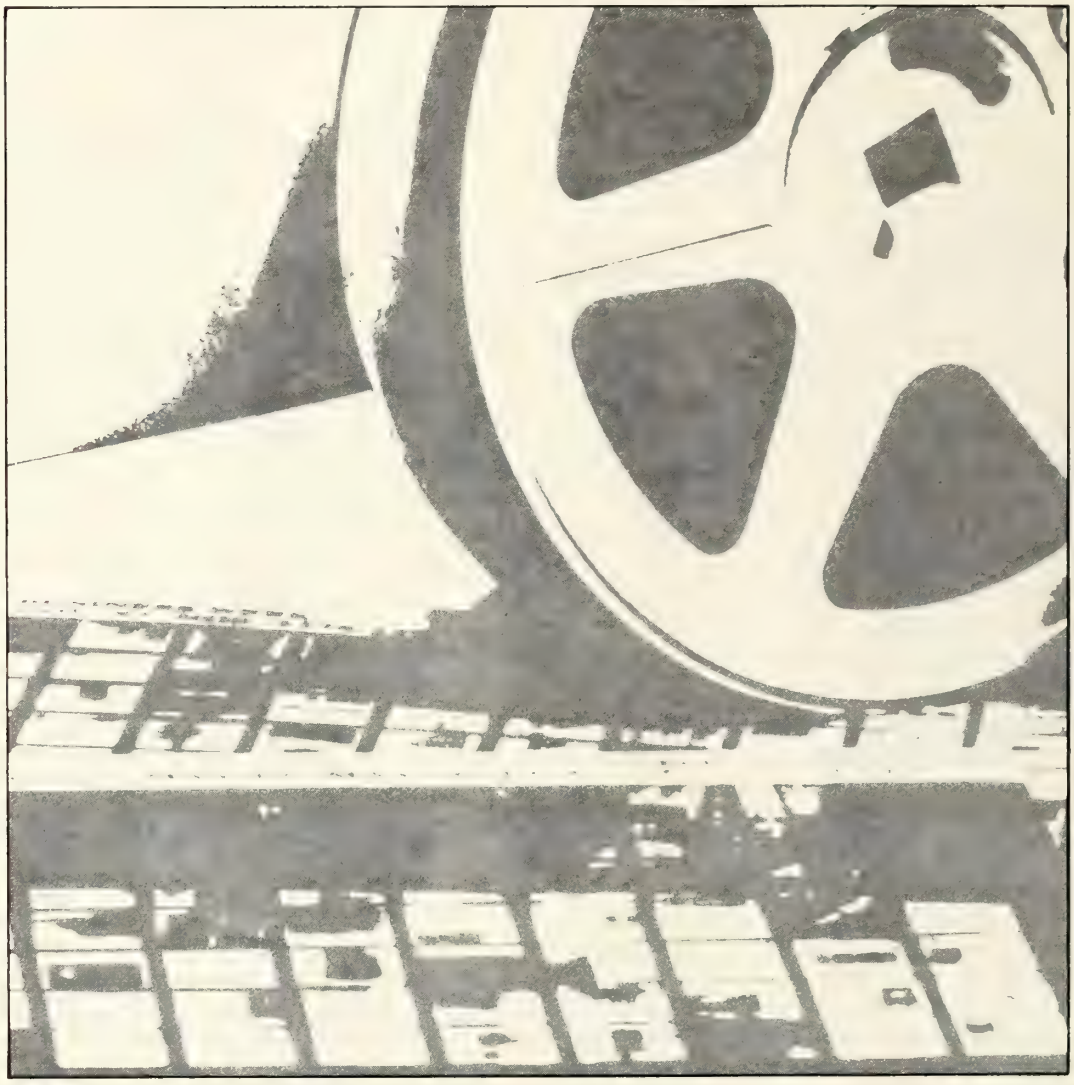

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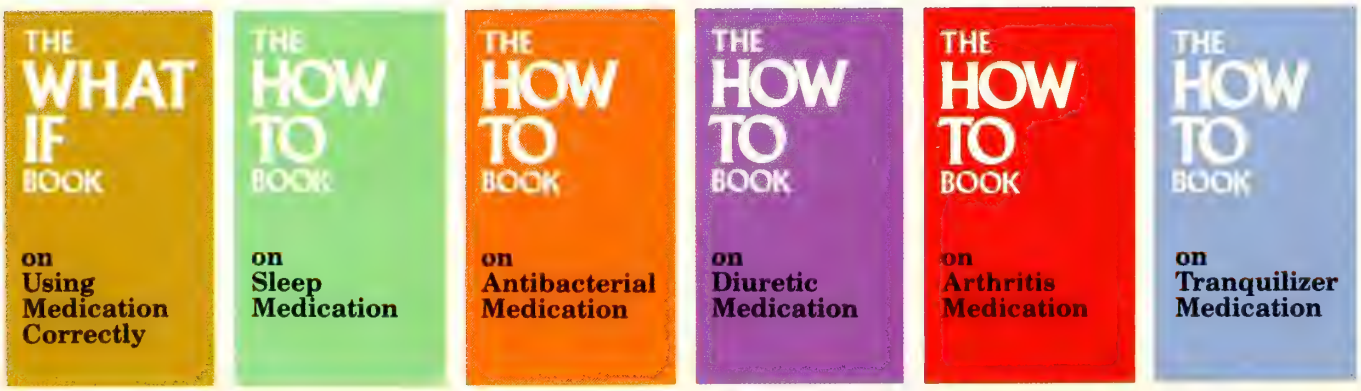
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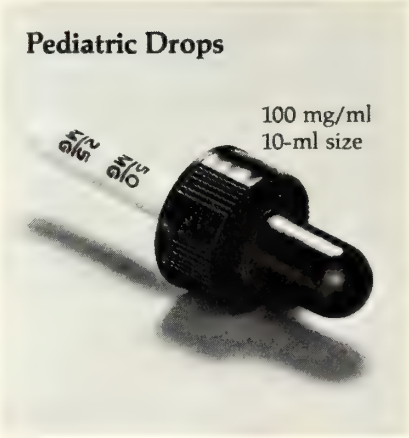

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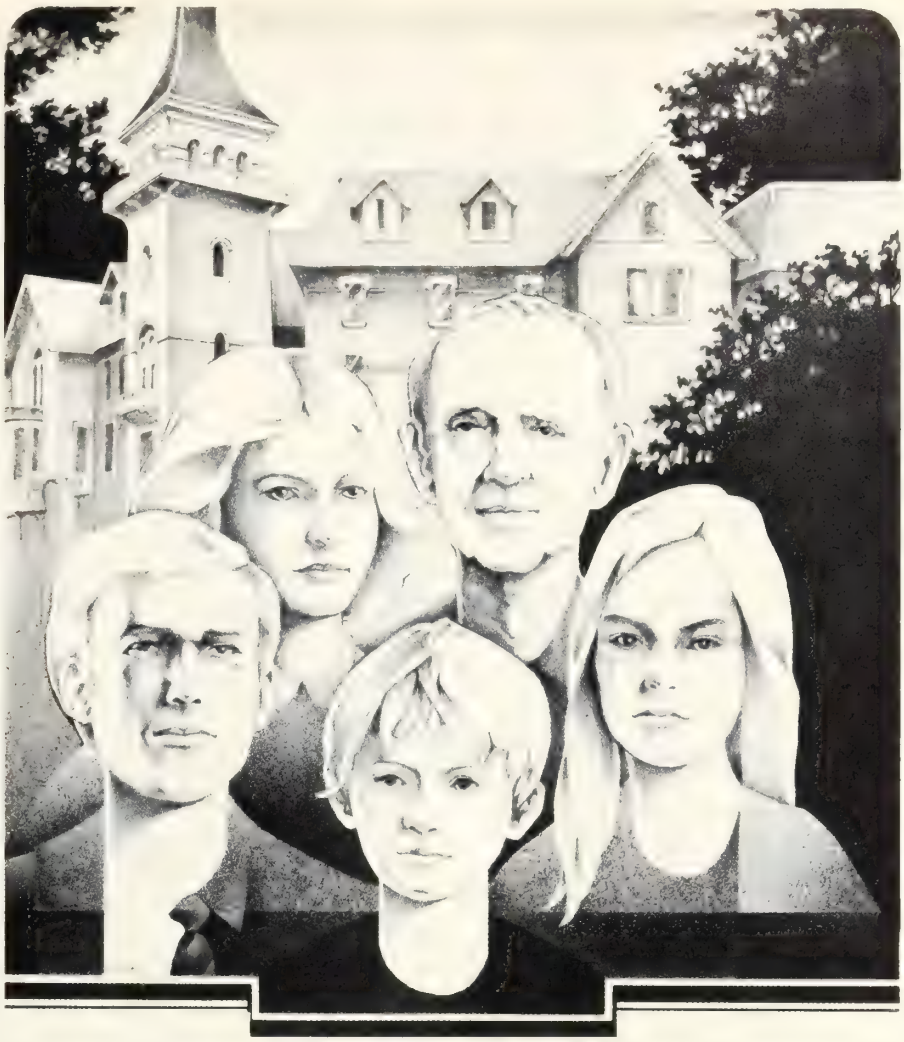

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VIRGINIA MEDICAL

EDITORIAL

The Strength of Idealism

"HEAL THE SICK, help the weak, care for the poor, free the captive, be true to your faith."[1] Emblazon this simple, magnificent declaration above our doors and in our minds! Or is it too idealistic? Is idealism dying?

"Heal the sick . . ." Certainly we can try, but this leaves no margin for the incurably ill, for the non-complier, for the self-destructive, for the litigious. In the courtroom the plaintiff's lawyer might ask, "Doctor, isn't there a statement on your wall which you say is your ideal?" "Yes," we reply. "Well, doctor, your patient died. You failed your own standards which you have emblazoned on the doorposts of your office. Sir, not only are you guilty of malpractice but also of misleading advertisement."

". . . help the weak . . ." Justice for all—weak or strong—is our legal ideal which has run amuck. The perpetrator is more protected than the victim. Traditionally we physicians have policed our ranks, sometimes more effectively than others, for the incompetent physician—whether ill, uninformed or dishonest. The unintentionally errant physician is usually penitent and seeks to change his ways. But the dishonest physician can hire enough lawyers to lodge enough appeals so that he may continue his fraudulent ways for years without loss of license or privilege. Voices roar for physicians to purge their ranks while simultaneously raising barriers to foil that process. Voices demand more continuing medical education for physicians while simultaneously granting medical privileges to those who never went to medical school and who possess little medical knowledge or skills. And the victim of all this obfuscation is not the well-informed but rather the weak and ill-informed patient.

". . . care for the poor . . ." For generations physicians did this without edicts and directions from the state and without red, white and blue cards. We reduced or eliminated fees for those whom we considered poor. But now we hear endless debate on the definition of poor. Some give away their assets to their children in order to become "officially poor" and therefore eligible for Medicaid, etc. And we physicians have endless forms to complete for those who continually reapply to Social Security for "disability status." The apellations "poor" and "disabled" are sought because they have distinct financial advantage.

". . . free the captive . . ." All Americans wanted the Iranian captives freed and rejoiced at their release. But what of the captives in South Vietnam, Cambodia, Africa, the Middle East, Russia? These people are captives in their own countries and cannot legally emigrate. And what of the captives in our country—free to move and to speak but captives of their environment and circumstances? Or what of the battered child who is returned to the abusing parents by a legal system with misplaced sympathy? Or what of those who are captive to their own prejudices and bigotries? They would be pitiful if they were not dangerous.

". . . be true to your faith." This could easily be misinterpreted as we slowly drift away from the constitutional ideals of separation of church and state and of freedom of religion. Today political

problems are couched in religious terms so as to cast the opposition "against the word of God." This nation must never be at the mercy of those few who would relay to us "God's words" without their publicly pondering why God should speak exclusively to them.

"Be true to your faith" means simply that. Your faith, your faith; my faith, my faith; and mutual respect and tolerance between us. We can all agree on standards of right and wrong, honesty and dishonesty. Everything is not relative. If you are rich, prominent or famous, you should have no more rights than the lowest of us. No longer should a theft of $5,000 bring a longer jail sentence than the theft of $5 million. And how do some elected officials become rich while in office? How do some religious leaders become opulent?

Idealism is not popular today. Cynicism, realism, opportunism and legalism threaten to displace idealism, while idealism without common sense is like a rocket without a guidance system. Yet great documents—the Bible, the Declaration of Independence, the Bill of Rights—reflect the strength of idealism. Nothing is wrong with idealism except that too few practice it. Rational idealism will never die because it is the guide to social progress. Cynicism, realism, opportunism and legalism should not and cannot abolish idealism. Temporarily outmoded though we rational idealists may be, we proudly proclaim: "Heal the sick, help the weak, care for the poor, free the captive, be true to your faith."

H. S. CAMPELL, MD

1. Gates of Prayer. Central Conference of American Rabbis, 1975 edition, p 277

Waiting for the Hundredth

In their article elsewhere in this issue, Drs. Centor and Gardner have captured well the current consensus of most diabetologists regarding tight control.

After the fruitless expenditure of a great deal of time and money, the University Group Diabetes Program, which originally set out to evaluate the results of tight control, ended up without a definitive evaluation of anything. Meanwhile, the animal studies cited by the authors come as close to fulfilling Koch's postulates as any we know—at least as regards diabetic glomerulopathy. They seem to show that hyperglycemia per se is the initial step in the pathogenesis of the diabetic complication. The literature is bursting with reports of abnormal glycosylation of proteinaceous components of the microvascular tree, in association with sustained hyperglycemia. With the appearance of preliminary reports of clinical improvement—or at least delay in deterioration of diabetic complications—in patients who achieve tight control, the argument would appear to be all over. Indeed, as the authors say, "There is no case for 'loose' control."

After this paper was submitted, a presentation at the May 1982 meeting of the American Federation for Clinical Research provoked considerable soul-searching. Tamborlane and associates at Yale reported that 17 Type I diabetics, enjoying tight control for 18-24 months by means of subcutaneous insulin delivered continuously by pump administration, did not demonstrate a single example of convincing improvement in microvascular disease. Rather, four of 24 eyes showed progression of retinopathy; proteinuria showed a transient decline, then returned almost to previous levels; serum creatinine rose minimally; and glomerular filtration rate fell minimally (Clin Res 30:405A, 1982).

What does it all mean? Perhaps very little. Even 17 patients is not a large number, and two years is certainly not long enough to be convinced of a definite and progressive trend or non-trend—even though the changes in experimental animal tissues have occurred much sooner.

Furthermore, there is no denying the relative power of a positive experiment. Even though 99 studies may show a thing to be impossible, they are all swept away and consigned to the attic of outmoded science with the advent of the hundredth study that proves, for the first time, that the thing is possible.

Does this mean then that we can ignore Tamborlane et al? Not at all, for the final proof, the hundredth study, has not yet appeared. If and when it does, it will in all likelihood be designed very much as Tamborlane et al designed theirs, using the most advanced technological methods and repeatedly confirming successful control by unfailing doc-

umentation of normal blood glucose and glycosylated hemoglobin. And yet—if Tamborlane et al could maintain tight control for two years in 17 diabetics, without the slightest improvement in microvascular complications, is there any point in repeating *that* experiment, using those techniques, at the present time?

The whole question reduces itself to the basic dilemma of all research: When is a thing finally proven? When should we cease to be excited about inventing the wheel and begin to be bored with reinventing the wheel?

The answer is clear. We must continue to press for proof that the wheel has in fact been invented, is reproducible, and is generally available. We must not let anticipation run away with scholarly judgment.

The crucial role of tight control in management of diabetic complications may well be said to be proven—in animal studies. Logically, we deduce that the same benefits would proceed from the same interventions in human diabetes. It may be so, but analogy is not proof, and as scientists we cannot safely say at this time that such a deduction is justified.

Inventing the wheel, then, still lies before us, and it may well prove a formidable undertaking until we can arm ourselves with something more effective than even the newest open-loop external devices that are now coming on the market. We may have to wait for the totally implantable closed-loop artificial pancreas or, more likely, for a solution to the problems which currently prevent islet-cell transplantation. Then, and only then, can the Tamborlane study be repeated with a reasonable expectation of positive results. And when those results emerge, as in all probability they will, what a glorious triumph for the millions of diabetics in this country alone, not to mention the growing numbers all over the world! It will be like Banting and Best all over again, staying up night after night with their diabetic dogs that long hot summer in Toronto over 60 years ago. . .

So long as we are all agreed that such a time has not yet arrived, it is still entirely fitting and proper to proceed just as Drs. Centor and Gardner would have us proceed—diligently striving to help our patients achieve the tightest control possible in the most consistent fashion possible, day in and day out, year in and year out, until The Day arrives. We must be careful not to promise too much, lacking final proof. But while we wait for it, the art of medicine requires that we act on faith, and "faith is the substance of things hoped for, the evidence of things not seen."

JOHN A. OWEN, JR., MD

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University of Virginia School of Medicine
Charlottesville VA 22908

"Make your voice loud and clear"

WHAT makes this such an exciting opportunity to speak to you is the chance to tell you about the wonderful things so many people have been doing to make our Society such a strong, vibrant organization. The real work of the Society is done by these people, who work diligently and without recognition.

I would like first to mention the work of Dr. Charles Crowder's Membership Committee, because membership is the life blood of our Society. As that committee report indicates, this past year has been a tremendous effort on multiple levels toward increasing our membership. What better way to strengthen the services of our Society or to delay that inevitable increase in dues than to recruit new members? One thousand new members means $150,000 more with which to expand our services and finance our programs.

Over the past years, our efforts through membership committees, VIRGINIA MEDICAL, the work of officers and councilors, and direct mailings to nonmembers has been satisfying to a degree only. From this point on, it seems to me, it is simply up to each individual one of us by direct personal contact. We have a tremendous amount of ammunition available to us as reflected by the agenda for this House of Delegates, the committee reports, and the many things that I am going to be able to speak about in the next few minutes that have been accomplished by you yourselves, and we have a new weapon in the form of our Membership Directory.

Delivered as the President's Address before the House of Delegates at the annual meeting of The Medical Society of Virginia on November 11, 1982, at the Williamsburg Conference Center.

After conferring with Dr. Williams, he and I would like to make this proposal to you. Inside your package you have some membership application blanks. We ask that between now and our annual meeting at the Homestead next November you recruit four new members. That would be well over our 1,000 new members. Dr. Williams has agreed that at the next annual meeting, he will ask all of those who bring in four new members to stand and be recognized.

I recognize next the hard work of Dr. Charles Caravati's Bylaws Committee, the results of which are before you. I know that you will give your serious attention and thought to their important recommendations regarding both the Articles of Incorporation and the Bylaws.

One of the shortest reports, which to my mind makes it the biggest understatement of the year, comes from the Insurance Committee, led by Dr. Alvin Connor. What makes this committee's work so important is that it relates to the most tangible benefit of belonging to the Society. If you are able to show a physician that he can save money through participation in any of our insurance programs, then you have made a great stride in recruiting that physician. This committee has done a superb job in negotiating our malpractice insurance, but the most trying area in the past year has been that of health insurance, most particularly, outpatient psychiatric coverage.

This subject has been covered fully in the past, so I will simply urge you all to scrutinize your health insurance programs from all sources very carefully over the next few months and please do not hesitate to make inquiries at our headquarters about specifics regarding our plan. This is an all-important recruitment tool, and we owe a rousing vote of thanks to our Insurance Committee for keeping it truly intact.

A whole new phase in our Society's work toward containing the costs of medical care is Dr. Edward Zakaib's Business-Medicine Coalition Committee. Time and again, during my year as your President, I have encountered questions about rising medical costs and what we physicians are doing about them, and I have been able to use this strong positive program as a most effective response. You can do the same in your membership recruitment efforts.

Another major weapon in recruitment is Dr. Glenn Updike's Legislative Committee, which has had another successful year on Capitol Hill. Allen Goolsby and Will Osburn contributed such outstanding expertise and direction to our program this year that the legislature gave us a .600+ batting average by agreeing with us 43 times in their actions on specific measures. That's a pretty good showing, and it has got to be one of our strongest recruiting tools since it is directed almost entirely toward the betterment of medical care in Virginia.

An item coming before the House of Delegates this year has to do with establishing a commission system to improve communication between our committees and the Council. Dr. Hagood and his committee have worked out the details of this concept for your consideration. I believe that this commission system will give more recognition to committees and their work between annual meetings and, in the overall sense, increase the efficiency and effectiveness of our Society.

The Vanguard Committee is before you in several ways at this annual session. I think we must commend our Vanguard Committee for the great job it has done over the years and for the national recognition it has brought us. I think that we all agree that our Society needs a committee to deal with federal legislation and governmental activities. I leave it to the wisdom of this House of Delegates to determine what form that will take, but I would like to stress that it should have full fiscal and staff support at headquarters, certainly equal to that accorded the Society's Legislative Committee.

Because of legislation passed under the Carter Administration, we physicians are often caught in the middle between Social Security and our patients, and Dr. McCausland's Rehabilitation Committee is right there in the middle with us. I urge you to visit the Disability Determination Services' booth here at this meeting, to enhance your awareness and knowledge of the dilemma facing our disabled patients.

I would be remiss not to mention Dr. Tony Munoz and his Scholarship Committee for completing a job well done. Our three medical schools now are giving justified recognition to the Society's support of medical education.

One of the truly exciting events of the past year and a great new weapon in our recruiting arsenal is the Physicians' Health and Effectiveness program developed by Dr. William Barney's committee. Please visit this committee's exhibit, and I urge your affirmation of the program presented in this committee's report.

The Education Committee, led by Dr. Eugene Temple as chairman and Dr. Kinloch Nelson as the Society's Director of Continuing Medical Education, has come out with a highly beneficial program of malpractice seminars designed to improve communication between physician and patient and be-

tween physician and hospital staff, all of which goes to strengthen patient compliance and understanding and improves medical care in general, while at the same time stemming the tide of litigation. These seminars are yet another recruiting tool.

It is with great pride that I point to the role that our journal, VIRGINIA MEDICAL, continues to play in our Society. We are greatly indebted to Dr. Edwin L. Kendig and his editorial board for the way our journal continues to present the scientific activity of our members through the many superb articles expressing their research and clinical studies. Ann Gray, the Managing Editor, continues to put together vibrant issues. We are fortunate to have her professional talent.

The complexion of VIRGINIA MEDICAL is changing to meet today's needs by becoming the major means of communication between you and your leadership. Dr. Kendig has been most gracious in accommodating to this new role, and Mrs. Gray has been most effective in promoting it with eye-catching presentations. This teamwork is strengthening the readership of our journal.

It has been a special pleasure to work with Mrs. Marguerite Hannon, President of The Medical Society of Virginia Auxiliary. To me, the Auxiliary is one of the greatest resources we have because of its public education program, which presents such a positive image of our profession to so many communities throughout Virginia. We should applaud and support in every way possible the many things the Auxiliary does to benefit our patients.

To the many contributions to the Society of Ed DeBolt and Carole Cones can be added not only this year's congressional meeting, which was a tremendous success, but a host of other highly important activities in the vital public relations area. Surely one of the greatest milestones in the history of this organization was when the Society joined forces with Mr. DEBOLT and his group. Each year the contributions differ, because the problems change, but I feel certain that this relationship will grow and become closer and richer as years pass.

This year, as expected, I have had the opportunity to observe firsthand the diligent work of our 12 staff members at Society headquarters in Richmond, one of the smallest staffs of major state societies in the country. To paraphrase Sir Winston Churchill, never have so few served so many so well. I am proud of the way these fine people have responded to our every need and request, and I feel that it is most important we in turn support them in their needs. There certainly could not be a more deserving group of people.

You cannot imagine the pride I feel as the first President to serve with James L. Moore, Jr., who became our Executive Vice President early this year. Jim's performance during this initial year can best be described as a continuance of excellent traditions but with some quiet innovations. Certainly this has to have been one of the smoothest administrative transitions that ever occurred, and the Long-Planning Committee is to be complimented for its selection of this splendid leader. I feel confident that you will exercise this same wisdom by reelecting him to the executive vice-presidency.

One of my greatest pleasures as your President has been the chairing of the meetings of our Council. It is truly the most sophisticated and effective group I know in carrying out its responsibilities, and I am sure you join me in my deep gratitude to them for a first-rate job.

This year I have learned so much about the wonderful work each of you is doing to improve the quality of health and medical care in Virginia. Importantly, I have received distinct impressions that you are leaving the physician's historical low profile in your community and speaking out strongly on behalf of our profession. To me, this represents a tremendous psychological step forward in lifting the bushel off our light, so to speak, revealing the many good things we do.

Now I would like to take a few moments to pass on to you some of the impressions and feelings that I have about our profession and about you and me that I have thought about as I have traveled the highways of Virginia this year. To me, one of the great challenges that we face this coming year is rising medical care costs. If you look at this past election you can see that the public is deeply concerned about recession, about costs, about balancing the federal budget, and I think the message to us as physicians is on the same subject. I think your Society is concerned, I think you are concerned, and I think what we have been doing this year reflects that concern.

We have heard a lot of talk about the physician glut and the effect this has on competition. To me the critical point about competition is that we not look at this in the commercial sense, which is to increase the demand for goods and services by increasing the unit consumption, at the same time decreasing the price to generate profit. How we should approach competition is by improving the services to our patient, improving our own professional skills.

HADDON CHRISTOPHER ALEXANDER, III, MD,
PRESIDENT, 1981–1982, THE MEDICAL SOCIETY OF VIRGINIA

Photograph by Susan C. Alexander

We have so many other challenges to face us in the future. I think we have to face the challenge of the hospitals because of the pressures on them regarding costs. We are aware that every review of utilization shows that hospitals are an area of greatest cost. We contribute 20%, but the rest of the arena of medical care costs to the gross national product is 80%. And everywhere a limit has been put on rising costs, it has affected hospitals. I think hospital administrators have faced this challenge of rising costs by changing their role—rather than return to operating in the red, they feel they must operate as business executives and keep their institutions operating in the black. I think all these things have to bear on our profession.

The people are concerned about us and our profession and our ability to police ourselves. We have certainly made tremendous efforts in these areas. We have tissue committees, review committees, and many things delineated in our hospital staff bylaws aimed at this. The real problem, we all know, is that our colleague we're reviewing is entitled to the same rights as anyone else. This is called due process. And in exercising these rights and protecting this individual we run into the problems that a police officer might have in reading his rights to a criminal. There are definite limits of things we can do not to intrude on the rights and privileges of the individual person.

I'd like to focus down on the things that I feel are the very essence of our profession, that of our relationship to our patient. The patient wants privacy, he wants intimacy, he wants one-on-one contact with us. If you think about the physical examination setting, never is anyone in our society more unprotected, figuratively and literally bared, than in that situation. And our patient wants to have the intimacy and privacy of the one-on-one relationship with his physician. I think that as long as we reflect on our every move and action that we definitely have great concern, deep empathy, and deep willingness to help him, that our patient is going to be our protector. Our patient is going to be our guardian angel provided we heed his wants and recognize his needs and, to me, that is the thing we should use in our principal, guiding philosophy in all the problems we face in these coming years.

When you look at the community, it simply is a collection of more patients and more physicians just as ourselves in different groups. Patients tend to group together in economic strata, the private-pay patient, the insurance-covered patient, the indigent patient, but all with similar concerns. When you look at us, we physicians group together as specialists. And this is because we need to have a grip on the immense amount of scientific data. On the other hand, in order to deal with all the institutions we have to face, and this is a concept that I borrowed from Dr. Jack Taylor in his inaugural speech before the Norfolk Academy of Medicine, we have to face social structures that are heavyweights, institutions. In the socioeconomic arena, as specialists we do not have the clout and numbers that are necessary. What we have to have is the power and ability of collectiveness and concerted efforts to take on these institutions we have to face—institutions of local government, institutions of federal agencies, institutions of the third-party industry, and of anything else you want to mention. If you look around at who it is with whom we have to deal in these situations, it's institutions, and I challenge you to look at your organized medicine system as a series of institutions beginning, as Dr. Jirka said, with the grass roots of your component society, moving on up to your state society and the work you've done, on up the national level at AMA.

To me, the word for this philosopy is sociocentric, that sociocentric philosophy in which our patient comes first, and in anything we endeavor to do that we apply this measuring rod to it; if it is good for our patient, it is good for us. If it serves his needs, if it supports the quality of medical care, then it's the thing for us to be doing.

THERE are many urgent matters to be dealt with at this meeting of our House of Delegates. What a great help and support it was, during the past year, to have the guidance of the House's actions at last year's meetings in Norfolk. Accordingly, I urge you to give the same thought and consideration to these pressing matters as our delegates always have done in the past Your President, your Council, and your headquarters staff need your mandate to guide them through the year. Yours is the responsibility to be the voice of the membership, and I urge you to make this voice as loud and clear as you possibly can.

Being your President this past year is the greatest privilege I have ever had, and I know nothing will ever replace its premier place in my life. With deepest and most heartfelt appreciation, I thank you.

H. C. ALEXANDER III, MD,
President, 1981-1982,
The Medical Society of Virginia

VIRGINIA MEDICAL OBITUARY

Robert R. Eason, MD

Dr. Robert Richards Eason, general practitioner in Buena Vista for 45 years, died October 22, 1982, in Stonewall Jackson Hospital, Lexington. He was 75 years old.

Dr. Eason was graduated from Randolph Macon College in 1931 and in 1936 received his medical degree from the Medical College of Virginia. His long-time family practice and service as chief of staff at Stonewall Jackson Hospital earned him recognition as Buena Vista's Outstanding Citizen of the Year in 1977.

For 44 years a member of The Medical Society of Virginia, Dr. Eason was also a member of the Rockbridge County Medical Society, Southern Medical Association and American Medical Association.

Memoir of J. Edwin Wood 1897-1982

By Lockhart B. McGuire, MD, John A. Owen, MD and John L. Guerrant, MD

Dr. J. Edwin Wood, Jr., exemplary physician and stimulating teacher to several generations at the University of Virginia School of Medicine, died at his home on July 26, 1982.

Dr. Wood's energy and eagerness for the science of medicine were evident in his early training. After receiving the MD degree at the University of Virginia in 1921, he took additional training at the Massachusetts General Hospital in Boston until 1923. There he began research in the circulation and electrocardiography with Dr. Paul White, and from 1926 to 1927, he continued this work in London as a National Research Council Fellow in the esteemed laboratory of Sir Thomas Lewis. He returned to the medical faculty in Charlottesville, became professor of internal medicine in 1936, and directed activities in both clinical and experimental cardiology there for 31 years until his retirement in 1967. His aptitude for study and experiment led to numerous publications and distinctions. In addition, his membership in the national community of physician-scientists during a period of limited support and time for such activity provided an important base for the subsequent expansion of clinical cardiology, surgery, and research at the university. During these years Dr. Wood provided much valuable leadership in the faculty. Later he was a member of the staff of Martha Jefferson Hospital.

Dr. Wood's reputation as a diagnostician, personal physician, and bedside teacher exceeded his contributions as a scientist. He enjoyed teaching medical students, helping them distinguish the important from the trivial, reduce the complex to the straightforward, and encouraging them to see beyond the immediate and obvious dimensions of a subject. He was an exciting lecturer, who could keep his audience awake after lunch on a warm, spring day.

Dr. Wood introduced or made familiar in Virginia numerous diagnostic or therapeutic procedures, including the electrocardiogram, fluoroscopic examination of the heart, a clinically useful method for measuring serum electrolytes, the use of mercurial diuretics for heart failure and the treatment of sepsis by a sulfonamide.

In great demand as a consultant, Dr. Wood often made trips to other communities to see especially ill patients. Fortunate indeed was the student or house officer who was invited to go along in his sporty red car to "carry his bags," record the electrocardiogram or take a blood sample, but more important, to observe him at work and listen to him during the ride. Legions of grateful patients appreciated his capacity for concern, wisdom, and optimism. Emphasizing the conservatism born of great experience, he was willing to be innovative and aggressive at appropriate times.

Dr. Wood's other enthusiasms included his family, tennis, and fly-fishing with his friends. He had the satisfaction of seeing his son, J. Edwin Wood III, become an eminent professor of medicine, and his grandson a recent graduate in medicine. He is also survived by his wife, Emily Battle Wood; a daughter, Mrs. William Corey Trench, and several grandchildren.

His career brings to mind a statement of Osler: "To study the phenomena of disease without books is to sail an uncharted sea, while to study books without patients is not to go to sea at all."

WHO'S WHO

Following in the footsteps of his father, **Dr. M. Pinson Neal, Jr.,** Richmond, was installed as president of the Southern Medical Association at its 76th Annual Scientific Assembly in Atlanta. The late Dr. Neal Senior, a graduate of the Medical College of Virginia and for many years professor of pathology at the University of Missouri, was SMA president in 1942.

Educated at the University of Missouri and the University of Tennessee School of Medicine, Dr. Neal Junior interned at the Medical College of Virginia and joined MCV's Department of Radiology in 1963. He has been chairman of the Division of Diagnostic Radiology and assistant dean of the School of Medicine, and has led as president the Virginia Council on Health and Medical Care.

For the Southern Medical Association, Dr. Neal has served in many appointive and elective offices. Founded 75 years ago, the association now has a membership of 27,000 physicians from 16 southern states and the District of Columbia.

Named president elect of the Southern Medical Association Auxiliary at the same meeting was **Mrs. William J. Reardon,** whose husband is a family practitioner in McLean. An illustrious figure in state and national auxiliary leadership, Mrs. Reardon has been president of The Medical Society of Virginia Auxiliary, national chairman of the American Medical Association's Education Research Foundation, and vice president for the AMA Auxiliary's Eastern Region. She will be installed as president at the 1983 meeting in Baltimore.

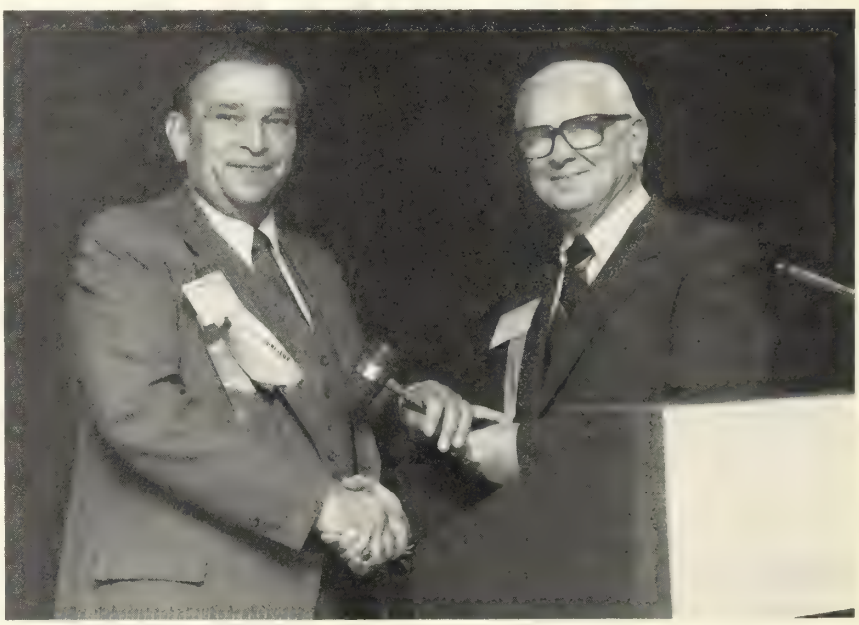
To honor Dr. Neal and Mrs. Reardon, The Medical Society of Virginia gave a reception during the SMA meeting at Atlanta's Peachtree Plaza. In the Virginia contingent were **Dr. H. C. Alexander III,** Roanoke, then MSV President, and **Dr. George J. Carroll,** Suffolk, himself a past president of the Southern Medical Association.

A Past President of The Medical Society of Virginia, **Dr. James P. King,** was honored for 50 years of service to Saint Albans Psychiatric Hospital, Radford, at a recent meeting of the hospital's board of directors. Dr. King is the son of the late John C. King, MD, who founded Saint Albans in 1916. He joined his father at the hospital in 1931 and was its president and medical director when he retired in 1972.

At the same meeting, **Dr. William D. Keck** was elected president of the Saint Albans staff.

Dr. Kevin R. Cooper, Richmond, was named Outstanding Volunteer of 1982 by the Richmond Region, American Lung Association of Virginia. Cited was his role in the passage of a local ordinance restricting leaf burning, his active support of the national Clean Air Act and his opposition to the relaxation of federal carbon monoxide regulations. Dr. Cooper is a member of the Medical College of Virginia faculty.

Patients of **Dr. Clarence J. Weimer,** Arlington, came to an open house last summer to honor him on his retirement. A general practitioner, Dr. Weimer was graduated from the Johns Hopkins School of Medicine, trained at



Hail and farewell: New President Neal takes the gavel from Edwin C. Evans, MD.

Hopkins, then served in the United States Navy before opening his practice in Arlington in 1948.

New director of the Cumberland Plateau Health District is **Dr. Nellie Dorsey Wright,** Abingdon. A graduate of the Medical College of Virginia, Dr. Wright is a pediatrician.

For his "unprecedented accomplishments of service to his family, his profession, his church, and our community," **Dr. Robert L. A. Keeley** received the Distinguished Service Award of the Roanoke Academy of Medicine at a recent meeting of the Academy. A native of Roanoke, Dr. Keeley was graduated from the University of Virginia School of Medicine and performed his surgical residency at Duke University. The award was presented by **Dr. David P. Minichan, Jr.**

Dr. P. Declan Burke, Culpeper, has been elected to fellowship in the American College of Obstetrics and Gynecology—not, as was erroneously reported in these columns, the American College of Radiology, where he would most certainly be an ob-gyn out of context.

At fall convocation ceremonies in Charlottesville, **Dr. William H. Muller, Jr.,** was given the University of Virginia's highest honor, the Thomas Jefferson Award, for his accomplishments as "an uncommon scholar and a great man." A pioneer in heart surgery, Dr. Muller had been vice president for health affairs at the university since 1976 and, before relinquishing the post recently, was for 27 years chairman of the School of Medicine's Department of Surgery. The award is presented annually to an outstanding member of the university community.

Editor of what is called "the first comprehensive textbook on sports medicine" is **Dr. Daniel N. Kulund,** Charlottesville. Its title is "The Injured Athlete," it was released recently by J. B. Lippincott, and its 544 pages are illustrated by UVa artist Ronald J. Ervin. There are also photographs of athletes in action, from Virginia Cavaliers to weight lifters; many of these were taken by Dr. Kulund himself.

Dr. Kulund, professor of orthopedics, rehabilitation and physical education at the University of Virginia, prepared the book for health professionals, especially primary care physicians, who are seeing more and more sports injuries, but he hopes coaches and trainers will find it useful, too. Chapters describe such injuries as tennis elbow, torn knee cartilage and runners' bumps, and how to diagnose and treat them. One of the contributors is **Dr. Frank C. McCue III,** orthopedist and University of Virginia team physician.

In Lexington, there was a "**Dr. John Couper** Day" at Stonewall Jackson Hospital recently so that the hospital's directors, staff and employees could pay tribute to Dr. Couper on his retirement from the practice of anesthesiology at the hospital. Schooled at Virginia Military Institute and the University of Virginia School of Medicine, Dr. Couper first practiced in Englewood, New Jersey, before returning to Virginia in the early '70s to live and practice in Lexington.

His book a first: Dr. Kulund

In installation ceremonies at the Northern Neck Medical Association's meeting late last year, **Dr. John W. Johnson,** Kilmarnock, was installed as president and **Dr. Eugene J. Wolski,** Callao, as secretary-treasurer. Named president elect was **Dr. Carrington Williams, Jr.,** Kilmarnock, and as vice president, **Dr. Robert E. duPrey,** Kilmarnock.

In the secretarial post Dr. Wolski succeeds **Dr. Norman R. Tingle,** Lively, who had served in the post for 14 years; for both the verve and durability he had brought to the office, the membership commissioned a plaque of appreciation for Dr. Tingle, and **Dr. J. Motley Booker,** Lottsburg, presented it to him.

VIRGINIA MEDICAL CLASSIFIED

Virginia Medical classified ads accepted at the discretion of the Editor. Rates to Medical Society of Virginia members: $15 per insertion up to 50 words, 25¢ each additional word. To non-members: $30 per insertion up to 50 words, 25¢ each additional word. Deadline: 5th day of month prior to month of publication. Send to the Advertising Manager, 4205 Dover Road, Richmond VA 23221.

POSITION WANTED—Former Virginia resident finishing PHS obligation in West Virginia 7/83 desires position with established 3-4 member FP group. 1978 MCV grad, board certified FP. Especially interested Richmond to Valley region. Will consider multi-specialty group. Write Michael P. Taylor, MD, 1413 Ravina Road, Charleston WV 25314, or call after 6 PM (304) 345-4906.

FAMILY PHYSICIAN wanted for hospital affiliated general practice as long term locum at $70,000 per annum or partnership after one year, located in sunny southeastern Virginia, excellent recreational facilities and close proximity to urban center. Reply VIRGINIA MEDICAL, Box 64, 4205 Dover Road, Richmond VA 23221.

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NATIONAL GUARD—If you haven't thought seriously about the Virginia National Guard militia, you probably haven't thought seriously. For information, call or write: Joseph D. Brown III, MD, 224 Monticello Avenue, Williamsburg VA 23185, (804) 229-0765 (office) or 253-2532 (home).

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PHYSICIANS—We are looking for a Virginia-licensed physician to practice in our Radford Health Dept. Duties include examining, diagnosing and treating patients in Well-Baby, Family Planning, Maternity, and VD clinics and assisting in program development and health planning. Local travel throughout a multi-jurisdictional health district. We offer an 8-4:30 workday, allowing you weekends to enjoy the beautiful mountains of Virginia; a competitive salary ($37,092-$50,672), professional growth opportunities, and a generous fringe benefits package including malpractice insurance protection. INTERESTED? Submit Virginia State application or resume to Cynthia Shelton, Virginia Health Dept., Rm. 110 James Madison Bldg., 109 Governor St., Richmond VA 23219 by 5:00 pm, January 31, 1983. Equal Opportunity Employer.

RARE OPPORTUNITY to see/shop big, beautiful collection of antiques at the Richmond Academy of Medicine Auxiliary's 1983 Antiques Show and Sale. Dates: Friday to Sunday, February 18 to 20. Place: Science Museum of Virginia, 2500 W. Broad St., Richmond. Hours on Friday and Saturday, 10 am to 9 pm; on Sunday, 12 to 6 pm. For more information, call Mrs. Michael Jaffee, chairman, (804) 282-7372.

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References: 1. Kales A *et al: J Clin Pharmacol 17*:207-213, Apr 1977 and data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Kales A: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **3.** Zimmerman AM: *Curr Ther Res 13*:18-22, Jan 1971. **4.** Kales A *et al: JAMA 241*:1692-1695, Apr 20, 1979. **5.** Kales A, Scharf MB, Kales JD: *Science 201*:1039-1041, Sep 15, 1978. **6.** Kales A *et al: Clin Pharmacol Ther 19*:576-583, May 1976. **7.** Kales A, Kales JD: *Pharmacol Physicians 4*:1-6, Sep 1970. **8.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **9.** Dement WC *et al: Behav Med 5*:25-31, Oct 1978. **10.** Vogel GW: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **11.** Karacan I, Williams RL, Smith JR: The sleep laboratory in the investigation of sleep and sleep disturbances. Scientific exhibit at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971. **12.** Pollak CP, McGregor PA, Weitzman ED: The effects of flurazepam on daytime sleep after acute sleep-wake cycle reversal. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, June 30-July 4, 1975. **13.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ.

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Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

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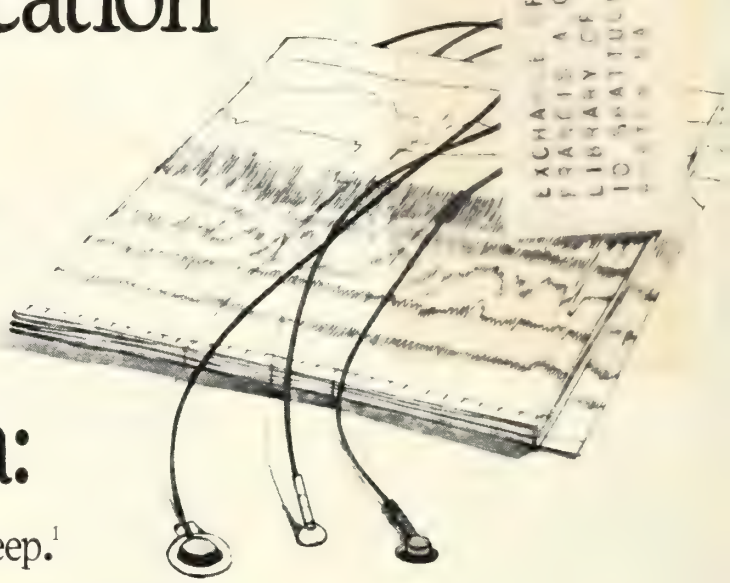
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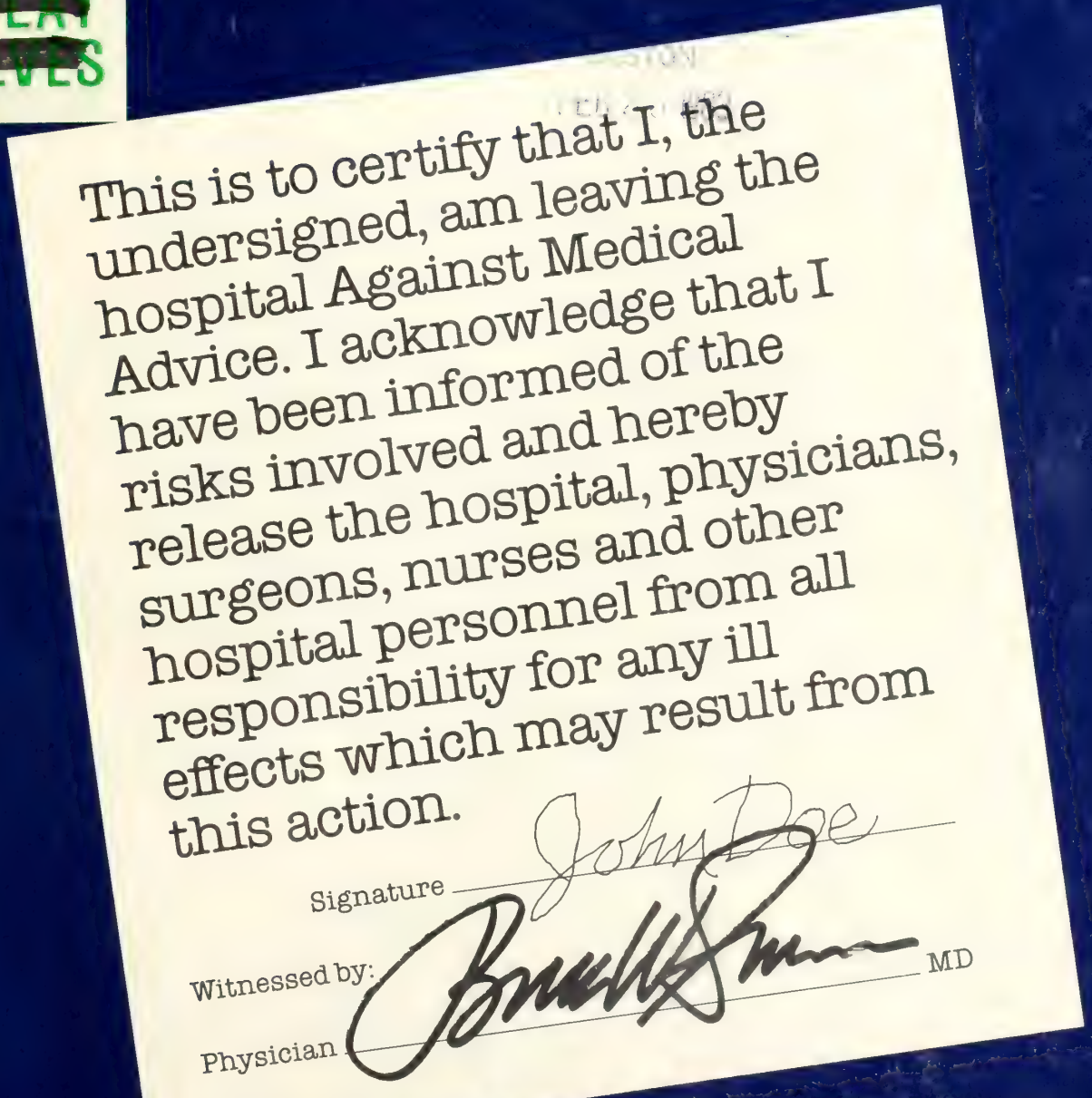

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Witnessed by:

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VIRGINIA MEDICAL

February 1983, Volume 110, Number 2

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LETTERS

Gives high marks to issue on "worrisome problem"

I simply wanted you to know that I found VIRGINIA MEDICAL's November issue on breast cancer and informed consent very outstanding.

I thought the unified concept and the diversity of the authors gave the whole problem of consent and management of breast disease an excellent and well-balanced presentation of a worrisome and controversial problem.

If future issues continue on this level, I am sure that you will be cited for outstanding medical journalism.

Thomas M. Wright, MD

7 Corners Professional Building
Falls Church VA 22044

What a difference a diphthong makes

It was with chagrin that we noted an old friend, Dr. James T. Tucker of Richmond, had died on July 14, 1982, at the age of 84. It was also very disturbing that the word "orthopaedic" was spelled incorrectly.

The improper spelling changes the meaning of the word orthopedic, which literally means straight foot. However, the correct spelling is orthopaedic—which means straight child. I think that in further issues it would be well to note that the spelling does make a difference in the meaning of the word and that it should be spelled correctly.

H. M. Kunkle, MD

3300 High Street
Portsmouth VA 23707

Empathy for Harvard history, but how to buy it?

I was delighted to see Dr. Grinnan's interesting review of Maxwell Finland's history of the Harvard Medical Unit at Boston City Hospital.[1] My intern days were spent on Four Medical, and I would like very much to have it. The review says it is distributed by the University Press of Virginia, but from whom do I order it?

W. T. Thompson, Jr., MD

4602 Sulgrave Road
Richmond VA 23221

1. Grinnan RB jr: Review of *The Harvard Medical Unit at Boston City Hospital, Vol. 1.* Va Med 109:819, 1982

Editors' Note: The book may be ordered from the Order Department, University Press of Virginia, Box 3608, University Station, Charlottesville VA 22903. To the price of $50, add $2 sales tax and $1 for postage. Or by donating $100 to Harvard University you will receive a copy gratis.

Warns other physicians of ersatz directory solicitation

In 1967, The Medical Society of Virginia issued a special service bulletin regarding the "classified directory problem," warning members that promoters were fraudulently soliciting physicians to buy space in classified directories, or "yellow pages."

I received one of these solicitations in the mail last summer from Classified Directory Publishing, Inc., Niagara Falls, New York, and sent a letter of complaint about it to the Postmaster General and the Niagara Falls postmaster. Recently I received a reply from the postal inspector in Buffalo, New York, as follows:

The firm [Classified Directory Publishing] has entered into two separate consent agreements relative to the mailing of these solicitations.

1) Title 39, United States Code, Section 2001(d) was amended because of this firm's mailings in 1978. A disclaimer is required which appears in bold green type and states: "This is not a bill. This is a solicitation. You are under no obligation to pay unless you accept this offer.

By the inclusion of this disclaimer on the invoice mailed by the firm, the firm is not violating federal law. This decision was reached by the United States

Attorney's Office, Western Judicial District of New York, and attorneys for the Postal Inspection Service's Law Department, Washington DC.

2) The second consent agreement was signed by the firm in September 1982. The firm agreed not to send renewal invoices to any firm that had previously paid the invoice unless they had paid for inclusion in the directory the previous year. The disclaimer as described is not required on these "one-year renewal" solicitations.

Your complaint will be forwarded to Classified Directory and they will remove your name from their mailing list.

I feel certain that other physicians are receiving these solicitations and write this letter in the hope it may warn members of The Medical Society of Virginia that this problem is a continuing one.

Peter A. Wallenborn, Jr., MD

201 McClanahan Street, SW
Roanoke VA 24014

Sees female "evolution" as path to toxic shock

Much scientific literature has arisen regarding "toxic shock syndrome," which has served to deemphasize the obvious predisposing cause—blockage of usual drainage from body cavities. Woman has "evolved" toward the wearing of tight pants and tampons, thereby altering the physiology so that, due to heat retention and lack of drainage, the basic pathophysiology for the development of toxic shock syndrome is created. Return to characteristic feminine attire and external padding has completely relieved many patients who previously have run the therapeutic gamut without improvement. To paraphrase, "The use of tampons may be dangerous to your health," and they should so be labeled.

Bernard Notes, MD

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10,000,000 alcoholics. Ethanol may produce many effects that together bring about nutritional deficiencies, so that alcoholism affects nutrition at many levels.[1]

25,500,000 geriatric patients. The older patient may have some disorder or socio-economic problem that can undermine good nutrition.[2]

23,500,000 surgical patients. Nutritional status can be compromised by the trauma of surgery; and some operations interfere with the ingestion, digestion and absorption of food.[3]

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Precautions: *General:* Certain conditions may require additional nutritional supplementation. During pregnancy, supplementation with vitamin D and calcium may be required. Not intended for treatment of severe specific deficiencies. *Information for the Patient:* Toxic reactions have been reported with injudicious use of certain vitamins and minerals. Urge patients to follow specific dosage instructions. Keep out of reach of children. *Drug and Treatment Interactions:* As little as 5 mg pyridoxine daily can decrease the efficacy of levodopa in the treatment of parkinsonism. Not recommended for patients undergoing such therapy.
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5,000,000 hospital patients with infections.[4] Many are anorectic and may have a markedly reduced food intake. Supplements are often provided as a prudent measure because the vitamin status of critically ill patients cannot be readily determined.[3]

The incalculable millions on calorie-reduced diets. Patients ingesting 1000 or fewer calories per day could be at high risk because this intake may not supply most nutrients in adequate amounts without supplementation.[5]

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References: 1. Shaw S, Lieber CS: Nutrition and alcoholism, chap. 40, in *Modern Nutrition in Health and Disease*, edited by Goodhart RS, Shils ME. Philadelphia, Lea & Febiger, 1980, pp. 1220, 1237. 2. Watkin DM: Nutrition for the aging and the aged, chap. 28, in *Modern Nutrition in Health and Disease, op. cit.*, p. 781. 3. Shils ME, Randall HT: Diet and nutrition in the care of the surgical patient, chap. 36, in *Modern Nutrition in Health and Disease, op. cit.*, pp. 1084, 1089, 1114. 4. Dixon RE: *Ann Intern Med 89* (Part 2): 749-753, Nov 1978. 5. Committee on Dietary Allowances, National Research Council: Recommended Dietary Allowances, ed 9. Washington, National Academy of Sciences, 1980, p. 13.

minerals, but generally at levels substantially higher than those in Berocca Plus. However, allergic and idiosyncratic reactions are possible at lower levels. Iron, even at the usual recommended levels, has been associated with gastrointestinal intolerance in some patients.

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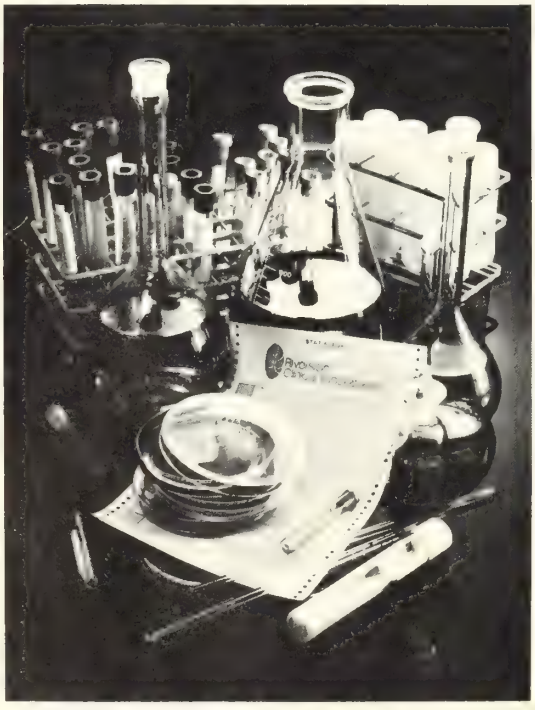

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- Measured against post-episiotomy pain in 30 patients, "ibuprofen was effective in treating the swelling as well as pain...during the first and worst days. Therefore, it is not only the analgesic but also the anti-inflammatory effect of ibuprofen that are the beneficial factors..."[4]

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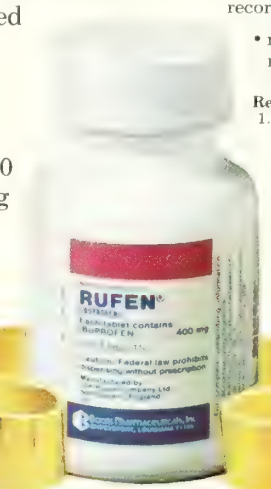

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4. Taina E: Curr Med Res Opinion, 7:423-428, 1981.

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INDICATIONS AND USAGE: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in the long-term management of these diseases. Safety and effectiveness have not been established for Functional Class IV rheumatoid arthritis.

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CONTRAINDICATIONS: Patients hypersensitive to ibuprofen, or with the syndrome of nasal polyps, angio-edema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory drugs (see WARNINGS)

WARNINGS: Anaphylactoid reactions have occurred in patients hypersensitive to aspirin (see CONTRAINDICATIONS). Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Peptic ulceration, perforation, or gastrointestinal bleeding can end fatally, however, an association has not been established. Rufen should be given under close supervision to patients with a history of upper gastrointestinal tract disease, and only after consulting the ADVERSE REACTIONS

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be attempted. If Rufen must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding

PRECAUTIONS: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If developed, discontinue Rufen and administer an ophthalmologic examination

Fluid retention and edema have been associated with Rufen; caution should be used in patients with a history of cardiac decompensation.

Rufen can inhibit platelet aggregation and prolong bleeding time. Use with caution in patients with intrinsic coagulation defects and those taking anticoagulants

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy, this therapy should be tapered slowly when adding Rufen

DRUG INTERACTION: *Coumarin-type anticoagulants* The physician should be cautious when administering Rufen to patients on anticoagulants

Aspirin Concomitant use may decrease Rufen blood levels

PREGNANCY AND NURSING MOTHERS: Rufen should not be taken during pregnancy nor by nursing mothers

ADVERSE REACTIONS Incidence greater than 1% Gastrointestinal: The most frequent adverse reaction is gastrointestinal (4 to 16%). Includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence). Central Nervous System: dizziness*, headache, nervousness. Dermatologic: rash* (including maculopapular type), pruritus. Special Senses: tinnitus. Metabolic: decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS) *Incidence 3% to 9%.

Incidence less than 1 in 100 Gastrointestinal: gastric or duodenal ulcer with bleeding and/or perforation, hemorrhage, melena. Central Nervous System: depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma. Dermatologic: vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome and alopecia. *Special Senses:* hearing loss, amblyopia (blurred and/or diminished vision, scotomata and/or changes in color vision) [see PRECAUTIONS]. Hematologic: neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs' positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit. Cardiovascular: congestive heart failure in patients with marginal cardiac function, elevated blood pressure. Allergic: syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasms (see CONTRAINDICATIONS). Renal: acute renal failure in patients with preexisting significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria. Miscellaneous: dry eyes and mouth, gingival ulcers, rhinitis.

Causal relationship unknown Gastrointestinal: pancreatitis. Central Nervous System: paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri. Dermatologic: toxic epidermal necrolysis, photoallergic skin reactions. Special Senses: conjunctivitis, diplopia, optic neuritis. Hematologic: bleeding episodes. Allergic: serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis. Endocrine: gynecomastia, hypoglycemia. Cardiovascular: arrhythmias (sinus tachycardia, bradycardia, and palpitations). Renal: renal papillary necrosis.

OVERDOSAGE: Acute overdosage, the stomach should be emptied. Rufen is acidic and excreted in the urine, alkaline diuresis may benefit.

DOSAGE AND ADMINISTRATION: Rheumatoid arthritis and osteoarthritis, including flareups of chronic disease: Suggested dosage 400 mg t.i.d. or q.i.d

Dysmenorrhea: 400 mg every 4 hours as necessary

Mild to moderate pain 400 mg every 4 to 6 hours as necessary for the relief of pain. *Do not exceed 2,400 mg per day.*

CAUTION: Federal law prohibits dispensing without prescription

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Antiminth offers ease of administration and patient tolerance. "...when compared to the other single dose agents available, [Antiminth] has the advantage of being non-staining and may be better tolerated."[2]

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Antiminth is available as a pleasant tasting, caramel-flavored oral suspension. Effective in just

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In some parts of the world, large populations are afflicted with helminthic infections. Physicians in endemic areas have become experts on parasitic diseases—and have come to rely on Antiminth for the rapid cure of infestations. Antiminth is recommended as an agent of first choice for pinworm and roundworm by leading medical authorities.[3]

Warnings

Usage in Pregnancy Reproduction studies have been performed in animals and there was no evidence of propensity for harm to the fetus. The relevance to the human is not known.

There is no experience in pregnant women who have received this drug.

The drug has not been extensively studied in children under two years; therefore, in the treatment of children under the age of two years, the relative benefit/risk should be considered.

Precautions

Minor transient elevations of SGOT have occurred in a small percentage of patients. Therefore, this drug should be used with caution in patients with pre-existing liver dysfunction.

Adverse Reactions

The most frequently encountered adverse reactions are related to the gastrointestinal system.

Gastrointestinal and hepatic reactions: anorexia, nausea, vomiting, gastralgia, abdominal cramps, diarrhea and tenesmus, transient elevation of SGOT.

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Children and Adults Antiminth Oral Suspension (50 mg of pyrantel base/ml) should be administered in a single dose of 11 mg of pyrantel base per kg of body weight (or 5 mg/lb.); maximum total dose 1 gram. This corresponds to a simplified dosage regimen of 1 ml of Antiminth per 10 lb. of body weight. (One teaspoonful = 5 ml.)

Antiminth (pyrantel pamoate) Oral Suspension may be administered without regard to ingestion of food or time of day, and purging is not necessary prior to, during, or after therapy. It may be taken with milk or fruit juices.

References 1. Pitts NE, Migliardi JR: *Clinical Pediatrics* 13:87, 1974. **2.** Modell W: *Drugs of Choice* 1980-1981. C. V. Mosby Co., St. Louis, 1980, p. 362. **3.** Goodman LS, Gilman A: *The Pharmacologic Basis of Therapeutics,* 6th edition, MacMillan Publishing Co., Inc., New York, 1980, p. 1032.

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1. National Interagency Council on Smoking and Health. *The Smoking Digest* Progress Report on a Nation Kicking the Habit. 1977

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Contraindications: Hypersensitivity; use with other xanthines

Warnings: Status asthmaticus is a medical emergency. Excessive doses may be expected to be toxic

Usage in Pregnancy: Safe use in pregnancy has not been established; do not use in pregnant women unless the potential benefits outweigh the possible hazards

Precautions: Use with caution in patients with severe cardiac disease, hypertension, hyperthyroidism, acute myocardial injury, congestive heart failure, or peptic ulcer. Chronic high dosage is usually associated with gastrointestinal irritation.

Adverse Reactions:

Gastrointestinal—irritation, nausea, vomiting, epigastric pain, headache, hematemesis, diarrhea

Central Nervous System—stimulation, irritability, restlessness, insomnia, reflex hyperexcitability, muscle twitching, clonic and tonic generalized convulsions, agitation

Cardiovascular—palpitation, tachycardia, extrasystoles, flushing, marked hypotension, and circulatory failure.

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Renal—albuminuria, increased excretion of renal tubule and red blood cells.

Others—fever, dehydration

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Regional Meeting, American College of Physicians, *Williamsburg*. James M. Moss, MD, 1707 Osage Street, Alexandria VA 22302, (703) 998-6511.

February 21-24
Alton D. Brashear Course in Head and Neck Anatomy (Medical College of Virginia), *Richmond*. 40 credit hrs. Fee, $300. Hugo R. Seibel, MD, Box 709, MCV Station, Richmond VA 23298.

February 25
Psychobiology in the Family: The Role of Genetics in Psychiatry (Psychiatric Institute Foundation), *Washington DC*. Marie Giblin, 4460 MacArthur, NW, Washington DC 20007, (202) 467-4538.

March 6-10
Gastrointestinal Radiology: 19th Annual Post-Graduate Seminar (Medical College of Virginia), *Williamsburg*. 19½ Cat. I credits. Fee: MDs, $350; residents, $175. Sheri Rosner, Box 48, MCV Station, Richmond VA 23298.

March 9-12
Internal Medicine 1983 (University of North Carolina), *Chapel Hill, North Carolina*. Office of CME, 231 MacNider Building, Chapel Hill NC 27514.

March 10-12
Practical Cardiology for the Primary Care Physician (American Heart Association, Tidewater Chapter), *Williamsburg*. 13 Cat. I credits. Fee: $200. Maria Ammerman, 891 Norfolk Sq., Norfolk VA 23456, (804) 857-5961.

March 17-18
Spring Meeting of the Virginia Chapter, American College of Surgeons, Hyatt House, *Richmond*. George A. Parker, MD, 8915 Norwick Road, Richmond VA 23229.

March 19
Third Annual Seminar of Cancer Researchers in Virginia (American Cancer Society, Virginia Division and Medical College of Virginia Cancer Center), *Richmond*. W. L. Banks, Jr., PhD, Box 37, MCV/VCU Cancer Center, Richmond VA 23298, (804) 786-0771.

March 20-24
Regional Meeting, American College of Cardiology, *New Orleans*. W. D. Nelligan, MD, 9111 Old Georgetown Road, Bethesda MD 20814.

March 21-25
Second Annual Review of Critical Care Medicine. (Johns Hopkins University), *Baltimore*. Cat. I credits. Fee: $450. Noreen Javornik, Office of CME, 720 Rutland Avenue, Baltimore MD 21205.

March 24-26
Gynecologic Oncology (Johns Hopkins University), *Baltimore*. Cat. I credits. Fee: MDs, $300; residents, $150. Susan Bavaro, Office of CME, 720 Rutland Avenue, Baltimore MD 21205.

March 25-27
Audiovisual Symposium on Short Term Dynamic Psychotherapy (Virginia Institute of Short Term Dynamic Psychotherapy), *Charlottesville*. 21 Cat. I credits. Fee: $150. Jason Worchel, MD, 1932 Arlington Blvd., Charlottesville VA 22903, (804) 977-6644.

March 27
Sports Medicine for Women (Eastern Virginia Medical School), *Norfolk*. Lou Ann Hamer, EVMS, PO Box 1980, Norfolk VA 23501.

April 6
Annual Clinical Conference (Louise-Obici Memorial Hospital), *Suffolk*. Cat. I credits. PO Box 1100, Suffolk VA 23434, (703) 539-1511, ext. 2692.

April 7-8
Ninth Annual Symposium on Diagnosis and Treatment of Neoplastic Diseases (Johns Hopkins University), *Baltimore*. Diane Hexdinger, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-6046.

April 8-9
Clinical Gastroenterology for Primary Care Physicians (University of Maryland), *Baltimore*. Office of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

April 8-10
Acute Trauma, Diagnosis and Management (Medical College of Virginia), *Williamsburg*. Fee: $250. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

continued over

April 11-16
Pediatric Trends (Johns Hopkins University), *Baltimore*. 45 Cat. I credits. Edward M. Sills, MD, Johns Hopkins Hospital, Baltimore MD 21205.

April 13-15
First Annual Southeast Regional Gerontology Conference (Eastern Tennessee Baptist Hospital), *Knoxville, Tennessee*. Sheila Gordon, PO Box 1788, Knoxville TN 37901, (615) 632-5061/5707.

April 15-17
Clinical Concerns in Primary Care (Medical College of Virginia) *Williamsburg*. Fee: $195. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

April 17-20
Current Topics in Allergy and Clinical Immunology (American Academy of Allergy and Immunology/University of Michigan), *Williamsburg*. 10½ Cat. I credits. Fee: $225. Office of CME, Towsley Center, University of Michigan Medical School, Ann Arbor MI 48109, (313) 763-1400.

April 17-22
Operative Treatment of Fractures and Non-Unions (Johns Hopkins University), *Hot Springs*. 36 Cat. I credits. Fee: $685. Carlita Kearney, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-3168.

April 21-23
Diagnosis and Treatment of Common Human Tumors (American Cancer Society, Kentucky Division), *Lexington, Kentucky*. American Cancer Society, Kentucky Division, 1169 Eastern Parkway, Louisville KY 40217.

April 21-23
Sports Medicine and Fitness Symposium (Georgetown University), *Washington DC*. Cat. I credits. Robert P. Nirschl, MD, 3801 Fairfax Dr., Arlington VA 22203, (703) 525-8000/2000.

April 22-24
Arrhythmia and Cardiac Ischemia: Diagnosis and Management (International Medical Education Corporation), *Williamsburg*. Cat. I credits. Mary A. Schroeder, IMEC, 64 Inverness Dr., E., Englewood CO 80112, toll free: (800) 525-8651.

April 28-30
Emergency Medicine (Medical College of Virginia), *Williamsburg*. Fee: $195. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

April 28-30
Tutorials in Interventional Radiology and Digital Subtraction Angiography (Johns Hopkins University), *Baltimore*. Cat. I credits. Fee: $275. Carlita Kearney, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-3168.

April 29
Pain Symposium (University of Maryland), *Baltimore*. Office of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

April 29-May 1
Clinical Management of Coronary Disease and Dual Mode Management (International Medical Education Corporation), *Arlington*. Cat. I credits. Mary A. Schrorder, IMEC, 64 Inverness Dr., E., Englewood CO 80112, toll free: (800) 525-8651.

April 29-May 1
Springfest: Program in Pediatrics (Medical College of Virginia), *Williamsburg*. Fee: $150. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

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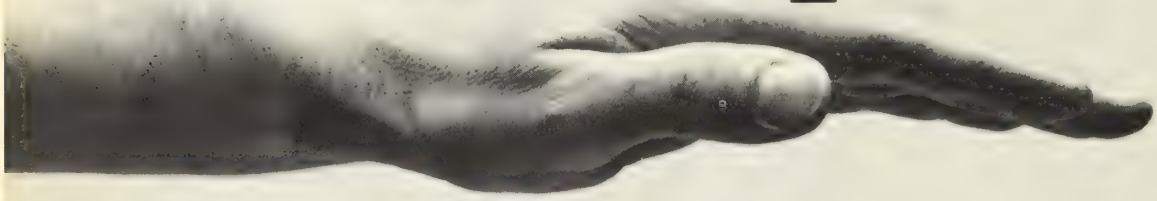

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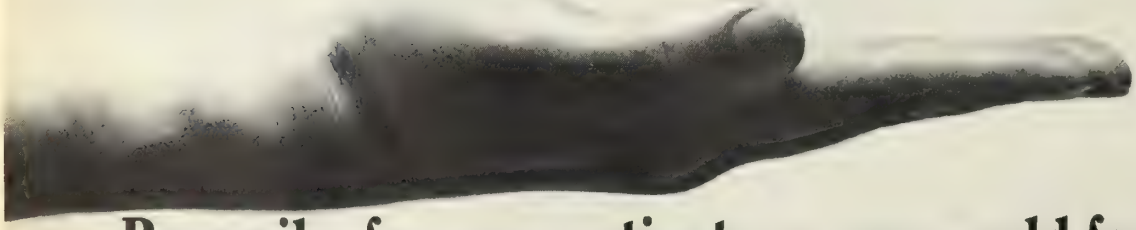

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Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

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Brief Summary. Consult the package literature for prescribing information

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms.

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci).

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions: *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip; USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor* (cefaclor, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below.

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

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*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.*

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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Leaving a Medical Service Against Advice

Kurt Link, MD, Charles E. Brody, MPH, and Jade Chan, PA,
Richmond, Virginia

An analysis of 57 patients who signed out of a university hospital AMA reveals that all patients were seriously ill and nine of them died within one year after discharge. The authors recommend that all hospitals have a followup protocol whereby the patient who insists on signing out is encouraged to return to treatment.

PATIENTS who leave the hospital against medical advice are a small but troubling group. Their decisions to leave, described as the "ultimate in non-compliance,"[1] dramatizes the inability to establish a therapeutic relationship and may produce concern, anxiety and anger in members of the hospital team.

Although there is a body of literature about patients who leave the hospital AMA, most of it pertains to psychiatric services, and there has been little attempt to document the subsequent clinical courses of these patients. There are four published series of these patients in whom the severity of illness was assessed. In two of these,[2,3] the patients who left against advice were found to be less severely ill than controls. In the other two,[4,5] the patients who left against advice were found to have serious illnesses.

To our knowledge, there is only one report that studies the courses of medical patients after they had left AMA; it showed that six of 31 patients who left a veterans administration hospital against advice died within six months.[5]

This two-year study follows the clinical courses of a group of patients who signed out AMA from the Medical College of Virginia Hospitals.

From the Division of General Medicine and Primary Care, Medical Collge of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Link at Box 285, MCV Station, Richmond VA 23298.

Presented in slightly different form at the Regional Conference of the American College of Physicians, Arlington, Virginia, February 25, 1982.

Methods

The charts of all patients who signed out AMA from the Medical College of Virginia Hospitals during the second half of 1979 were reviewed six months after they were discharged. If a patient had not returned to the institution within that time, we telephoned the patient or sent a letter of inquiry. If

there was no response, the procedure was repeated at six-month intervals during the two-year study. Data collected for each patient included demographics, diagnosis, clinical status and location of the followup medical care. If the patient or family could not be reached, the Bureau of Vital Statistics or coroner's office in the area of the patient's last known residence was contacted for any available information.

Results

During the last six months of 1979, 69 patients signed out against medical advice from the Medical College of Virginia Hospitals. This represented 2.8% of total medical discharges. Of these 69 patients, we determined the status of 57 (83%) between eight months and two years after discharge. Of these 57, nine (15.7%) had died. All of the deaths had occurred within one year after discharge, and five had occurred in the first six months after discharge.

We found that 23 (33%) of the total 69 patients who had left AMA had been admitted with chest pain known or suspected to be due to heart disease. We followed up 16 of these patients and found that four of them had died. In 11 (16%) of the total patients, the admitting diagnosis had been chronic obstructive pulmonary disease, asthma, or pneumonia. We followed up all of these patients and found there had been four deaths. The diagnosis of alcoholism or alcohol-related disease had been made in 20 patients (29%) of the total 69. We followed up 18 and found that one had died.

For the entire 69 patients who had left AMA, the average length of stay was 4.3 days; the average age was 54.8 years, and the proportion of males was 73%. For those who died, the average length of stay was 5.7 days, the average age was 46 years, and the proportion of males was 62%. For those not followed up, the average length of stay was 4.1 days, the average age 42 years; and the proportion of males, 67%.

Of the total group of patients who left AMA, 66% returned after discharge for further medical care and eight of the nine known deaths occurred in the Medical College of Virginia Hospitals, where the patients had previously signed out.

Discussion

The proportion of patients discharged AMA in our series (2.8%) is larger than that reported in two other studies (.7% and 1%)[2,6] but similar to that found at a veterans administration hospital (2.36%).[5] These differences may be related to the type of hospital, to demographic factors, or to the readiness or reluctance of the staff to allow patients to leave against medical advice.

In contrast to the findings of Schlaugh et al,[2] the patients in our study who left AMA were often seriously ill and had high six- and twelve-month fatality rates, especially those with heart or lung disease. The mortality rates for the group as a whole (14.5%) and the cardiac group (12%) cannot take into account any deaths that may have occurred in the patients lost to followup and therefore may be underestimated. On the other hand, these rates could be exaggerated if AMA discharge procedures were used selectively for very sick patients, but this is unlikely in view of our relatively high overall AMA discharge rate.

There have been numerous attempts to define some characteristics of the patient who is apt to sign out against medical advice.[6-8] These include alcoholism, drug addiction, anxiety and mental impairment. Unfortunately, these features are also prevalent in the acutely hospitalized patient, so they are of little or no usefulness in predicting which patients will sign out AMA, and efforts to anticipate and prevent an AMA discharge have not been successful.

As for the high mortality rate associated with AMA sign-outs, some strategy needs to be devised for affecting the clinical courses of these patients after their discharges. An informal survey of hospitals in the Richmond area suggests that most institutions do not have explicit policies regarding care after discharge for those who sign out AMA. The nursing and medical staffs involved in the patient's care generally try to dissuade him or her from signing out, but when the patient insists on leaving, followup plans are largely non-existent, and the return to the institution is left to the patient's initiative. Sometimes such a return is encouraged, but sometimes there are expressions of disinterest or outright hostility toward the patient.[9] Signing out AMA is regarded as a breach of contract, and the physicians of the medical staff often feel that patients who do so have no further right to medical care at the institution; it is as though these patients have relieved the institution of any further responsibility for their care.

Such an attitude can only exacerbate the serious consequences of leaving against medical advice, and to minimize these consequences, quite an opposite strategy is required. As soon as attempts to dissuade them are seen to be unsuccessful, patients who wish to sign out AMA should be offered a ready and rapid followup arrangement. The high

rate of return of these patients strongly suggests that such an offer would be accepted. Perhaps a team could be on call to respond whenever an AMA discharge seems imminent. Such a team might include nursing, general medicine, and psychiatric staff interested in this problem. We do not know if such a strategy would affect either the rate of these discharges or the rate of mortality following AMA discharge, but it certainly warrants a prospective study.

The authors are indebted to Ms. Maxine Burgess for her assistance in tracing patients.

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COMMENT

"Discharged against medical advice" is generally considered by practicing physicians as a black mark on a patient's record, indicating non-compliance and a troublesome doctor-patient relationship. As the paper from the Medical College of Virginia by Link et al in this issue of VIRGINIA MEDICAL indicates, however, many patients who reject medical care have serious illnesses and a high mortality rate, particularly if they are suffering from diseases of the heart and lungs. Many have problems with alcohol and drugs. It is important to note that more than half of the patients who signed out of the Medical College of Virginia Hospitals required further medical care at that institution, and eight of the nine known deaths in this group of patients occurred in subsequent hospitalizations at MCV.

These statistics point up the obvious: Sick patients are prone to behave in ways that make their care difficult. Compassionate physicians know this. But it is often difficult to be compassionate and understanding when faced with obstinance and rejection—"the ultimate in non-compliance." As physicians, we must make efforts to assure these patients prompt reentry into the system as soon as their turmoil subsides and they can accept proper medical care again.

The need for further medical care for these patients who sign out AMA has now been demonstrated beyond question. Our failure to assure such followup usually reflects our own hostility and anger, emotions which are not in character for those involved in the care of sick people. While it may be difficult to accept at the time of confrontation, these patients still require and should receive our best efforts.

The facts presented by Link et al will help me when I next am asked by the nursing staff what to do in these unpleasant situations.

THOMAS L. GORSUCH, MD

The Professional Center
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Cancer Trends: The Role of CEA in Managing Colorectal Cancer

Harold J. Wanebo, MD, *Charlottesville, Virginia*

Reviewing the experience with the carcinoembryonic antigen assay in the clinical management of colorectal cancer, the author finds instances of its usefulness as an indicator of recurrence, as a guide to second-look surgery, and as a preoperative signal in primary resection. He suggests continuing study of the assay's contribution to patient survival.

ALTHOUGH the carcinoembryonic antigen (CEA) is not specific for colorectal cancer, its strong correlation with many aspects of the disease has made it very useful in the clinical management of this malignancy. There is controversy, however, about the real value of CEA determinations in the clinical decision-making process and the impact on patient prognosis. Three major questions of concern are: Is the preoperative CEA of any value? Does CEA correlate adequately with the stage of disease and with different patterns of recurrence? Does serial CEA testing after surgical resection permit earlier detection of recurrence (than obtained on clinical grounds) and can one realistically select patients for second-look surgery on the basis of CEA elevation alone?

From the Department of Surgery, Division of Surgical Oncology, University of Virginia Medical Center, Charlottesville VA 22908.

The Cancer Trends series appears under the editorship of J. Shelton Horsley III, MD; Gerald Goldstein, MD; and Anas El-Mahdi, MD. It is sponsored by the Professional Education Committee, oirginia Division, American Cancer Society.

Submitted 4-29-82.

Carcinoembryonic antigen is a high molecular weight cell-surface glycoprotein first described by Gold and Friedman in 1965 as a unique component of human colon carcinoma and fetal colon tissue.[1] Two types of immunoassays for CEA are currently in use, a radioimmunoassay and an enzyme-linked immunosorbent assay. Radioimmunoassays use radioiodinated CEA, anti-CEA, and a precipitation method to separate bound from free antigen. The latter is usually accomplished by adsorption with zirconium phosphate gel[2] or other techniques such as a second antibody.[3] Some assays use sera[2] and others use plasma treated with perchloric acid (PCA). Enzyme-linked immunosorbent assays as developed by Hammarstrom[4] make use of binding of anti-CEA to plastic tubes and measurement of the competitive binding of CEA and anti-CEA antibody linked to an enzyme, alkaline phosphatase. The final product can be measured by colorimetry. Antigen is prepared from liver metastasis of colon cancer using either the perchloric acid extrac-

tion method or the neutral pH method of extraction. Commercial testing kits as provided by Hoffman LaRoche make use of a radioimmune assay of perchloric acid extracted antigen and precipitation by Z-gel method or as provided by Abbott Laboratories are made from an enzyme-linked immunosorbent assay using heat-precipitated antigen.

There are differences in sensitivity, precision, and the normal range between those two assays. It is essential for the physician to be familiar with the range of normals and the variation in the assay used in one's particular hospital setting. Because of variation, hospital laboratories may need to consult with the reference laboratories to assure quality control of the assay. Detailed information about the biochemical and immunological characteristics of CEA as well as other antigens that cross-react with CEA are provided in several reviews.[5–8] Because most of the data has been accumulated using the Hansen Z-gel method[9–10] of radioimmunoassay, we will concentrate here on those results. Although CEA levels of 0–2.5 ng/ml are commonly used as a normal cutoff point, some institutions consider 2.6–4.9 ng/ml borderline and values >5 ng/ml abnormal.[11]

It should be kept in mind that there are numerous causes of modest CEA elevation besides cancer, including inflammatory disease of the gastrointestinal tract and lungs,[12] alcoholic cirrhosis,[13] and smoking.[14,15] Approximately 15% of smokers (>15 cigarettes/day) may have elevations in the 5–10 ng/ml range, but rarely is smoking associated with elevations above 10 ng/ml. Higher CEA levels (>20 ng/ml) are very commonly associated with malignancy and usually with advanced disease. There is a fall-off in precision in the measurements of high values (requires dilution) but variation in this CEA range is of little practical consequence.

Preoperative Value

The incidence of abnormal CEA elevations varies according to stage of disease. If we use 5 ng/ml as a cutoff point, the preoperative CEA is elevated in 4% of patients with Dukes' A cancer; 26% of those with Dukes' B; 44% of those with Dukes' C; and 65% of those with Dukes' D cancer.[11] In data from the Memorial Sloan-Kettering Cancer Center, patients with Dukes' B or Dukes' C cancer who had preoperative levels >5 ng/ml, had significantly increased recurrence rates compared with their counterparts with CEA levels <5 ng/ml.[11,16–18]

Although clinicians are not accustomed to having this information available, knowledge of the preoperative CEA level could be of decided clinical usefulness. At the least, it might prompt the surgeon to be more assiduous in exploring the patient who is undergoing a resection for primary disease and to exclude the presence of disease beyond the primary site. Patients with preoperative CEA levels ≥20 ng/ml are prime candidates for liver metastases. A surgeon who is forewarned about the possibility of extracolonic disease might be more likely to identify the sites involved, which, in some cases, might be amenable to resection. It would be imperative in such cases to exclude any benign causes of CEA elevations, such as stones or inflammatory disease of the pancreas. If no extracolonic manifestations of cancer were found in a patient with a high preoperative CEA, such a patient would be considered at high risk for recurrence and might merit close observation with frequent postoperative CEAs. If the patient's CEA did not return to normal levels within 2-4 weeks, then very careful evaluation would be indicated, using the sophisticated radiologic techniques currently available, with consideration of an early second-look procedure. Although the surgeon rarely has the luxury of knowing the preoperative CEA, it would appear to offer a therapeutic advantage in at least some cases. To be useful, the preoperative assay would have to be rapidly done and reported to the clinician, which unfortunately has not been the case in the past.

Recurrence Indicator

Although there is controversy about the usefulness of CEA as an indicator of early recurrence,[15,18–24] there are distinctive correlations of CEA levels with patterns of recurrence. Over 90% of patients with metastases to liver, lung or bone have CEA levels >5 ng/ml, and 58-76% have levels >20 ng/ml. In contrast, only 50% of patients with local recurrence confined to the pelvis had values >5 ng/ml, and only 22% had values >20 ng/ml.[11] The highest CEA levels are obtained in patients with liver metastases, whereas in patients with pelvic recurrence, the CEA is frequently normal.[25–27] Moertel et al have claimed that the CEA is not a sensitive test for early recurrence of colorectal cancer.[18] In their series only 25% of patients with local recurrence in the pelvis had CEA levels >5 ng/ml. But CEA has been extremely useful in diagnosing recurrence at other sites, especially in the liver.

Obviously, this test must be combined with other clinical tests, the most important of which is a good history and physical examination. A history of pain in the perineum or posterior thigh following resection of rectal cancer is highly suggestive of recurrence. An awareness of symptoms and signs of

recurrence may potentiate earlier diagnosis. It is important to correlate clinical findings and impressions with CEA levels.

Mach et al have reported followup studies of 66 patients who had serial CEA levels over a five-year period after resection of colorectal cancer.[28,29] Of 19 who developed recurrence, 17 had increased CEA levels preceding clinical diagnosis by two to 26 months. Among 47 patients without evidence of recurrence, 35 had CEA levels remaining <5 ng/ml, whereas 12 had levels fluctuating around or above this level. Among these patients the history of smoking was prevalent and three had heavy alcohol intake. Because of these variations, this group did not consider patients who had CEA levels around 5 ng/ml for second-look surgery but repeated the CEA at least twice over a three-month period before making a decision.

In a contrasting study reported by Sugarbaker et al,[16] monthly CEA levels were done in 32 patients, of whom 12 developed recurrence. A rising CEA was the first evidence of recurrence in only four, and in these, all had distant metastasis. In a similar study Mackay et al,[30] measured CEA levels in 220 patients after curative resection of colorectal cancer; fifty-three patients developed recurrence and 36 of these showed a previous or simultaneous increase in CEA, but in 17 there was no change. An additional 17 patients had developed at least transient rises in CEA, but none had clinical evidence of disease. (None were explored, however.)

In a more positive study from Roswell Park, Herrera et al[31] followed 23 patients who developed recurrence and noted that 20 had had an elevated CEA at the time of clinically apparent recurrence, and in 14 patients the CEA was elevated by a mean of 5½ months prior to clinical evidence of disease. Neville and Cooper monitored sequential CEA levels in 82 patients, all of whom had experienced recurrence.[20] In 63 patients the CEA level elevation coincided with recurrence, and in 37 of these the CEA elevations preceded clinically detectable recurrence.[20] In 19 patients, however, the CEA was normal at the time of recurrence. Thus there were false-negative CEA values in about one-fourth of the patients. In the overall study from Memorial Hospital of 155 patients with documented recurrence, 28% had normal CEA levels.[11] A large proportion of these were patients with local recurrence in the pelvis.

Second-Look Surgery Indicator

The second-look procedure was first popularized by Wangensteen, Lewis and Tongen in 1951 to detect early recurrence after curative resection of colorectal cancer.[32] Although this approach was theoretically sound in the management of patients who are at high risk for recurrence, the procedure suffered because of the frequency of negative laparotomies, the frequent discovery of unresectable tumors, and the considerable morbidity and even mortality that ensued with a "routine" second-look operation.[32,33]

In 1972 Martin and Minton began using the CEA as an indicator for selective second-look surgery in patients who had had a previous curative resection of colorectal cancer.[34] In their first report in 1976 of a retrospective series of 22 patients who had had laparotomy because of an elevated CEA, recurrence was documented in 19 patients (80%).[35] The disease was localized in six (27%) and distant in 13 (59%). Six patients had resectable disease. The average CEA at the time of intervention was 7.2 ng/ml. In the subsequent "prospective series", CEA levels were obtained monthly, and recurrence was found in 17 of 18 patients (94%), and only one negative laparotomy occurred. In this series, the cancer was localized in 13 patients (72%) and the average delay from the awareness of elevated CEA to the second-look procedure was 1.4 months. The average CEA was 6.5 ng/ml in those with localized disease and 15.5 ng/ml in those with distant metastases.

A key point in Martin and Minton's evaluation was the use of a CEA nomogram constructed by preparing five different sample concentrations and assaying the duplicate sample 25 times to determine intraassay error. Interassay error was established by freezing aliquots of CEA containing samples of various concentrations and analyzing these over a three-month period. The 95% confidence limit was established for any given result, and the baseline established for each patient. If a patient's followup CEA was above the 95% confidence limit from the baseline, it was repeated. If a subsequent test was abnormal, both samples were reanalyzed. (In this assay the samples had been stored at −20°C.) If this assay confirmed an elevation above the 95% confidence level, a second-look procedure was then proposed. The ideal CEA followup according to these authors was monthly for two years; at two-month intervals for the next three years; semi-annually from the 5th to the 7th year; and annually thereafter.

There is controversy over this approach and concern that strict adherence to this nomogram would result in many unnecessary operations. Rittgers et al prepared a similar nomogram and

found 25 patients with CEA levels that met the Ohio State criteria for significance.[21] Nine of these patients had increases which were transient. Although one patient ultimately developed recurrence, the other eight had no clinical evidence of recurrent cancer, and might have had a negative second-look procedure if this had been recommended on the basis of CEA alone. In 16 (64%) of their patients, the CEA level rose progressively, and 13 of these had histologically documented recurrence. Of the three remaining patients, two had negative explorations and one very elderly patient was not considered an acceptable surgical candidate.[21] Thus there are areas of concern which need to be creafully examined before hard and fast rules can be applied. Although a nomogram can monitor test variability and document significant rises in CEA, it does not necessarily account for biologic causes, i.e., smoking, cholelithiasis, benign liver disease, etc.

A recent review has summarized the data from four institutions, comprising a total of 86 patients who had CEA-directed second-look surgery for possible recurrent colorectal cancer.[36] Recurrence was documented in 78 to 94% of the patients. The interval of testing ranged from approximately six to 12 weeks or longer. The average CEA prompting intervention ranged from 6.5 to 21 ng/ml, with much higher values recorded in individual series. The median delay from an abnormal test to surgery ranged from 4.5 to 5.3 months. Approximately 46% had local recurrence and 54% had distant metastases. Resection with curative intent was possible in 36%, ranging from 7%-72%. The variation among institutions was quite marked, indicating variation in the patient population, intensity of the utilization of the assay, and frequency of followup. The long-term results regarding survival are awaited with interest.

Because of the variation in results in the collected series, it is worth mentioning the current findings in the Ohio State program that pioneered the use of CEA-directed second-look surgery and has been most aggressive in applying it. In a recent update documenting their experience with a total of 60 second-look procedures, tumor was confirmed in 53 (93%).[37] The tumor was later confirmed in three of the four "negative" patients. Of 38 patients studied "prospectively" in this series, 60% had resectable tumor and 21 (35%) were free of disease from one to seven years.[37]

A more conservative approach was used in the Memorial Sloan Kettering experience, in which patients were generally explored on the basis of CEA elevations obtained every two or three months but without the vigorous use of the nomogram. The median CEA prompting second-look surgery was 21 ng/ml. In the initial reported series 14 of 16 patients were found to have recurrent tumor. Two had negative explorations (both had cholelithiasis and one of them developed recurrence later). Resection with curative intent was done in seven patients and four of these were surviving free of disease from 12–37 months at the time of that report.

In a recent update of the series by Attiyeh et al,[38] 32 patients have had second-look surgery and recurrence was documented in 28 of these; 16 patients had localized tumor, and in 13 a curative resection could be done.

It seems reasonable, then, that in most series of CEA-directed second-look procedures, disease will be found in 85%-90% of the patients explored, and in about 50% a resection can be done. This seems to be the case where even less vigorous use is made of the CEA test and explorations are carried out at CEA levels higher than that recommended by the Ohio State group, It is this author's contention that although the use of the nomogram as suggested by Minton and Martin is theoretically sound, there are problems with religious adherence to it as pointed out by Rittgers et al.[21] Perhaps a compromise would be to use the nomogram to document the occurrence of a significant rise, but then to take the precaution of searching for all benign causes of transient CEA elevations utilizing biliary tract studies and liver biopsy if necessary. Where there is no nomogram available, the clinician can still monitor CEA changes and make decisions based on these changes. Certainly a doubling of the CEA level, i.e., from 2.5 to 5.0 ng/ml, should prompt a repeat test. If the value continues to increase by another 50%, i.e., to 7.5 ng/ml and then to 10 ng/ml in the absence of smoking, this should prompt consideration of recurrence. Continued increases in CEA levels most strongly indicate recurrence and should prompt a complete diagnostic evaluation and probably a second-look procedure.

Effect on Survival

On the bottom line is the real question: What is the effect on patient survival? Only long-term data can establish this point. One is prompted to point out that there are several series in which patients who have had resection of solitary liver metastases from colorectal cancer have had survival rates ranging from 28 to 40%.[39–41] If the CEA assay can identify patients who have an early recurrence, such as a solitary metastasis to the liver or within the abdomen, these patients may be amenable to

resection and a potential cure may be accomplished. Although CEA-directed second-look surgery is theoretically appealing and has support from certain non-controlled surgical series, the true usefulness of CEA as a monitor to detect recurrence and increase patient survival can only be determined by a prospective randomized clinical trial.

Finally, CEA may have usefulness in patients with advanced disease as an indicator of response to chemotherapy or radiation therapy. Although this aspect has promise, more studies are needed to define the limits of CEA testing in this regard.

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Comment

Dr. Wanebo has extensively reviewed the place of the CEA assay in the management of colorectal cancer. Rising levels of CEA are of great value in indicating recurrence of carcinoma. Unfortunately, recurrence is not ruled out by normal levels of CEA. As long as one is not lulled to sleep by undue reliance on normal levels, information provided by the CEA test is very useful in the followup of carcinoma of the colon, and in monitoring the treatment of patients with metastatic or recurrent colon carcinoma.

GERALD GOLDSTEIN, MD

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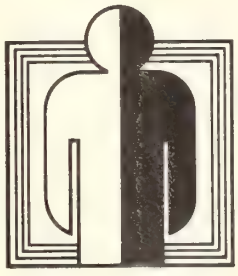

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Endoscopic Sphincterotomy to Remove Gallstones: Case Reports

Young S. Hong, MD, and Alan Harris, MD,
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Fig. 5. Extracted stone, Case 2, 1.5 × 1.0 cm.

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Endoscopic sphincterotomy was introduced in Japan and Germany as an investigative procedure in 1973 and has been performed in the United States for the last five years.[1,2] Although originally intended for the nonoperative removal of common bile duct stones at the time of endoscopic retrograde cholangio-pancreatogram in the post-cholecystectomy patient at high operative risk, indications for endoscopic sphincterotomy now include common bile duct stones in high risk patients with or without previous cholecystectomy, retained or recurrent common bile duct stones in healthy patients following cholecystectomy, and papillary stenosis.

In addition, obstruction to biliary and pancreatic ducts caused by periampullary tumors can be relieved by endoscopic sphincterotomy,[3] this palliation enabling definitive surgery after the patient's improvement. Endoscopic sphincterotomy is also of palliative benefit in the case of large common bile duct stones which cannot be extracted after a sphincterotomy incision of optimal length is made. (However, emergency drainage, allowing more thorough pre-operative preparation, can be obtained by the placement of a transnasal biliary catheter deep to the stones at the time of sphincterotomy.[4])

For relief of acute pancreatitis due to common bile duct stones, endoscopic sphincterotomy may be less dangerous than operative management, with its significant mortality, but since acute pancreatitis is normally considered a contraindication of endoscopic retrograde cholangio-pancreatography, this indication is not firmly established.

Endoscopic sphincterotomy is also occasionally performed for the drainage of choledochoceles.[5]

Absolute contraindications to endoscopic sphincterotomy are relatively few and include the presence of a long stricture beyond the intraduodenal portion of the common bile duct and significant coagulopathy. Relative contraindications are the presence of a stone larger than 2-2½ cm and acute pancreatitis.

Methods

Endoscopic sphincterotomy is performed with the use of fluoroscopy. The procedure begins with cannulation of the common bile duct using the side-viewing duodenoscope. Injection of radiopaque material defines the anatomy of the biliary and pancreatic ducts and demonstrates the location of common bile duct stones. The cannula is removed, and the specially designed sphincterotomy knife is placed through the scope (Fig. 1), selectively passed into

the distal common bile duct, advanced into the papilla until 2 to 3 mm of the exposed cutting wire remains visible.

Once the sphincterotome is in the proper position within the distal common bile duct (Fig. 2), the diathermy wire is pulled tense, thus forming the cutting bow, and an incision of 10-15 mm is made through the cephalad aspect of the sphincter between 11 and 12 o'clock. Sudden efflux of bile signifies decompression of the common bile duct. Injection of contrast material following sphincterotomy confirms the spontaneous passage of the stones or determines the location of the remaining stones.

About two-thirds of common bile duct stones can be expected to undergo spontaneous passage one to two weeks after successful sphincterotomy and can be recovered in the stool,[6] but the remaining must be extracted. We perform this extraction by means of the balloon catheter (Fig. 3) or the Dormia basket. We prefer the balloon catheter to avoid the risk of impaction of the stone.

Case Reports

These cases illustrate our initial experience in five patients undergoing endoscopic sphincterotomy for the extraction of biliary calculi.

Case 1. A 20-year-old female was admitted to the hospital because of right upper quadrant pain. She had cholecystectomy because of gallstones one month prior to admission. The laboratory study revealed elevated SGOT and SGPT of 320IU/L and 306IU/L respectively, elevated alkaline phosphatase of 430U/L and elevated total bilirubin of 2.2 mg/dl. Amylase was normal at 39IU/L. Endoscopic retrograde cholangio-pancreatography revealed one stone within the common bile duct about 0.7 cm in diameter.

Endoscopic sphincterotomy was done, direct extraction of the stone was accomplished with the balloon catheter, and the patient was discharged on the second hospital day. All of her liver function tests returned to normal in one month and she continues free of symptoms.

Case 2. A 68-year-old male who had cholecystectomy four years prior to admission was admitted to the hospital following the progressive onset of right upper quadrant abdominal pain and fever. Temperature on admission was 102.7 F, and WBC was 10,700/cu mm, SGOT and SGPT were somewhat elevated at 831U/L and 84IU/L respectively. Alkaline phosphatase was elevated at 148U/L and the total bilirubin was 5.1 mg/dl. Amylase was normal at 61IU/L. He was placed on gentamicin and cepha-

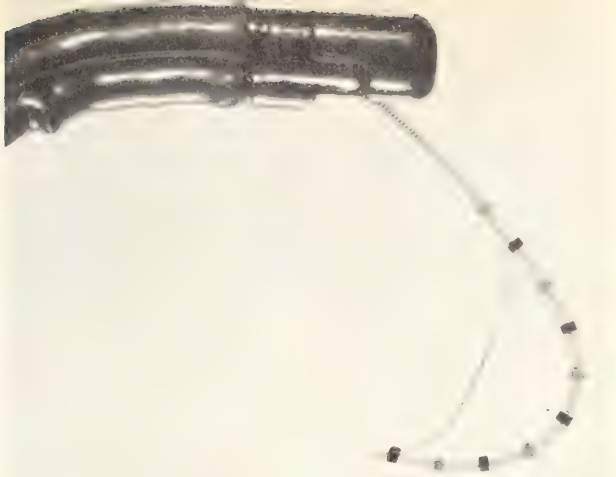

Fig. 1. Sphincterotomy knife advanced through side tip of duodenoscope.

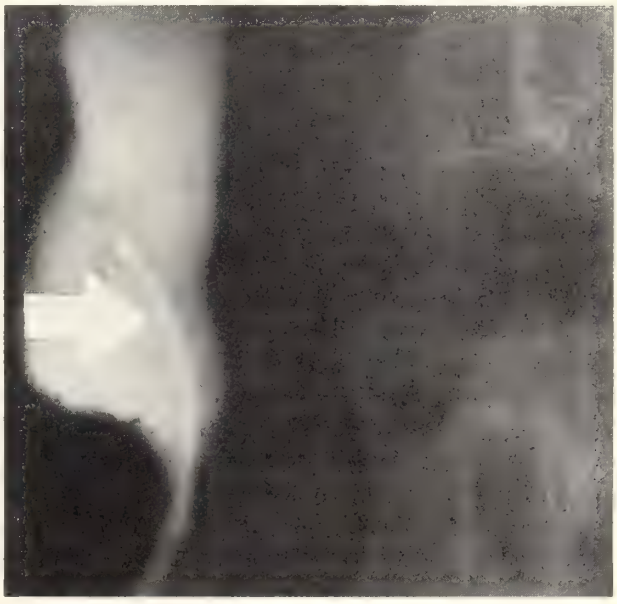

Fig. 2. Sphincterotomy knife placed in bile duct (arrow).

Fig. 3. Simulated stone extraction with balloon catheter.

Fig. 4. Stone (arrow) in common bile duct; pancreatic duct with beaded aspect (Case 2).

lexin. Endoscopic retrograde cholangio-pancreatography was performed and revealed a 1.5-cm common bile duct stone and pancreatic duct tortuosity consistent with chronic pancreatitis (Fig. 4). Since the patient had been treated for myasthenia gravis for ten years, endoscopic sphincterotomy was the preferred alternative to surgery. Endoscopic sphincterotomy was performed and the stone was extracted with the balloon catheter from the bile duct into the duodenum and was subsequently found in stool (Fig. 5). The patient was discharged three days later and remained asymptomatic.

Case 3. An 81-year-old female was hospitalized one month following cholecystectomy for gangrenous cholecystitis and cholelithiasis with the complaint of epigastric pain and persistent T-tube drainage. Liver function tests and amylase were within normal limits. A T-tube cholangiogram revealed a common bile duct stone one cm in diameter. Endoscopic sphincterotomy was performed, direct extraction with balloon catheter was attempted, but the stone was not seen to pass. When a cholangiogram failed to show the stone in the common bile duct, the patient was discharged on the following day. Repeat T-tube cholangiogram one week later, however, revealed a round filling defect within the common bile duct. The patient was readmitted, and the stone was expelled into the duodenum with a catheter through the T-tube tract.

Case 4. A 58-year-old female was admitted to the hospital complaining of severe epigastric pain two years after a cholecystectomy. She was febrile and liver function tests were elevated. SGOT was 1005IU/L and SGPT was greater than 300IU/L. Alkaline phosphatase was 165U/L and total bilirubin was 3.1 mg/dl. A diagnosis of ascending cholangitis was made, and she was placed on cefoxitin.

Endoscopic retrograde cholangio-pancreatography revealed two common bile duct stones. She underwent endoscopic sphincterotomy, with direct balloon catheter extraction of the two stones. Afterwards, she developed severe diarrhea and fever, and sigmoidoscopy revealed hyperemic mucosa. Stool was positive for Clostridium difficile toxin. Pseudomembranous colitis was diagnosed and she was placed on vancomycin with prompt improvement. She was discharged on the 10th day following the endoscopic sphincterotomy.

Case 5. A 61-year-old man was admitted because of nausea and vomiting. He had a long history of alcohol-associated hepatic cirrhosis and had undergone successful end-to-side portocaval shunt and resection of the caudate lobe of the liver because of massive hematemesis eight years previously. On this admission, liver function tests were SGOT 156IU/L, SGPT 130IU/L, alkaline phosphatase 435U/L, total bilirubin 9.7 mg/dl and serum ammonia 105 mcg/dl. Chest X-ray revealed significant bilateral pleural effusion. The patient was a very poor candidate for surgery because of extensive liver failure and poor pulmonary status. Endoscopic retrograde cholangio-pancreatography revealed several common bile duct stones ranging from 0.5 to 1.0 cm in diameter. Endoscopic spincterotomy was performed with no complications. Direct extraction with the balloon catheter was performed. Subsequent endoscopic cholangiogram revealed no filling defects. The patient's condition continued to deteriorate, however, and he died 17 days later because of hepatic failure and pulmonary infection.

Discussion

The utilization of endoscopic sphincterotomy has been steadily increasing since its introduction eight years ago. This is not surprising considering the advantages of endoscopic sphincterotomy, especially in the elderly and in high-risk patients. It is a brief procedure, taking only 30-90 minutes. Convalescent time is short, with total hospitalization ranging from 2-14 days.[11] Endoscopic sphincterotomy eliminates the need for general anesthesia, laparotomy and duodenotomy and therefore eliminates the prolonged recuperation period associated with surgery.

The reported overall complication rate for endoscopic sphincterotomy is 7%, including bleeding,

local perforation, pancreatitis, cholangitis, and stone impaction[7,8]; 25% of these complications require surgery. Complications are usually seen within a few hours following the procedure. Late complications are rare.

Followup reports have been encouraging. In Europe, five-year followup revealed restenosis in only 1% of patients and recurrence of common bile duct stones in only 2%.

The perioperative mortality of surgical sphincterotomy in patients older than 50 years was reported by Ackeren et al[9] and Bohmig et al[10] to be as high as 8.6%. But the perioperative mortality attributed to endoscopic sphincterotomy was reported by Koch et al[7] to be 0.8% in 254 patients and by Safrany[8] to be 1.2% in 243 patients, and all these patients were considered to be at high risk for surgery.

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ABSTRACTS

These are abstracts of papers to be presented at the 1983 regional meeting of the Virginia Chapter, American College of Physicians, in conjunction with the Virginia Society of Internal Medicine, on February 18-20 in Williamsburg. Dr. Jason E. McClellan is chairman.

Malnutrition in the Elderly: Role of Intestinal Bacterial Overgrowth. M. S. Wheby, MD, R. W. Lindsay, MD, D. D. Stone, MD, and R. L. Guerrant, MD, *Charlottesville*.

Bacterial overgrowth in the small intestine may cause a malabsorption syndrome. This may occur in the elderly as a result of altered intestinal motility with aging. Furthermore, new findings about colonizing and enterotoxigenic traits may be relevant to small bowel colonization and malabsorption. To investigate this possible relationship, we studied 18 people over age 65; 11 were well nourished while seven were considered malnourished on the basis of weight height relationships and anthropometric measurements. Studies include evaluation of: protein, vitamin, and mineral status; quantitative fecal fat determination, d-xylose tolerance and vitamin B12 absorption. All patients had jejunal intubation with quantitative bacterial cultures, ten of which included anaerobic cultures.

Of the 11 well nourished patients, three had $> 10^3$ coliform, anaerobic or neisserial bacteria/ml; while six of seven malnourished patients had $> 10^3$ coliform, anaerobic, staphylococcal, streptococcal or neisserial organisms/ml in jejunal aspirate. Unlike an additional patient with malabsorption and ileocecal resection whose 10^{10} *E. coli/ml* exhibited type I pili, none of the *E. coli* present in either study group had recognized adherence traits. One malnourished patient with malabsorption had 2×10^3 non-enterotoxigenic *Hafnia* sp/ml; the other had $> 10^7$ α streptococci and staphylococci/ml.

These studies indicate that malnutrition in the elderly may be associated with small bowel bacterial overgrowth that need not cause malabsorption. Bacterial attachment traits or enterotoxin production may be required for some organisms to cause malabsorption, and thus warrant further study.

Massive Gastrointestinal Hemorrhage Secondary to Rectal Ulcers in Patients with Chronic Renal Failure. Marvin Goldberg, MD, George C. Hoffman, MD, and Duane G. Wombolt, MD, *Norfolk*.

Massive lower GI bleeding from rectal ulcers is an unusual medical problem. The syndrome has never been described as a complication of chronic renal failure. We will discuss five patients with chronic renal failure who presented with a sudden onset of life-threatening hemorrhage secondary to solitary rectal ulcers. Four of the five patients were being treated with dialysis.

All of the patients had massive gastrointestinal hemorrhage with a passage of large amounts of bright red blood per rectum. Hypotension was present in all and none of the patients had been dialyzed within 24 hours of the bleeding and thus were not anticoagulated with heparin. All patients required transfusions. Two patients were considered for colectomy to stop the bleeding prior to the proper diagnosis. Two of the patients had mesentric arteriograms to help localize the source of bleeding. Three of the five patients had to be taken to the operating room for suture ligation to stop their bleeding. Biopsy of one ulcer revealed nonspecific inflammatory cells. It is important to consider the diagnosis of bleeding rectal ulcers in any patient with chronic renal failure who develops a sudden massive life-threatening lower gastrointestinal hemorrhage.

Improved Global and Regional Left Ventricular Function Following Successful Percutaneous Transluminal Coronary Angioplasty in Patients with Unstable Angina Pectoris. George Vetrovec, MD, Eric Carlson, MD, Timothy Wolfgang, MD, and Michael Cowley, MD, *Richmond*.

To assess the effect of unstable angina (UA) and subsequent improved coronary perfusion on global and regional left ventricular function, 30° right anterior oblique left ventriculograms were performed immediately pre and post successful coronary angioplasty (PTCA) in 10 patients (pts) with UA and severe single vessel disease confined to the left anterior descending (6 pts) or right coronary artery (4 pts). UA included recurrent rest pain and/or recently accelerated minimal activity pain. Similar pre and post PTCA left ventricular analyses were also performed in 8 pts with stable angina (SA). Mean vessel stenosis decreased from $92 \pm 2\%$ (SEM) pre to $41 \pm 3\%$ ($p \leq 0.05$) post PTCA for UA

and 95 ± 2% pre to 52 ± 9% post for SA. Global ejection fraction increased from 47 ± 5% to 62 ± 4% ($p \leq 0.05$) for UA pts but did not change (59 ± 4% pre, 61 ± 3% post, P = NS) for SA. Likewise, segmental ejection fraction (SEF) (segmental area method) in UA pts. showed a significantly ($p \leq 0.001$) greater increase (24 ± 4% to 47 ± 3%, $p \leq 0.001$) for the ischemic related segments than for the non-ischemic segments (39 ± 3% vs 50 ± 3%, $p \leq 0.01$). In SA, SEF did not change for ischemic segments (38 ± 3% vs 42 ± 3%, P = NS) or non-ischemic segments (45 ± 3% vs 48 ± 2%, P = NS).

In conclusion, successful PTCA is associated with acute improvement in resting global and regional left ventricular function in patients with UA.

Fibrinogen White Marsh—A New Human Fibrinogen Variant With Aα Chain Defect. G. D. Qureshi, MD, Herbert Evans, PhD, R. Marcus Vennart, MD, Joseph P. Magnant, BS, Joan M. Sabau, BS, and Jean Willoughby, BS, *Richmond.*

Congenital dysfibrinogenemia is a rare disorder of hemostasis in which structural and biochemical abnormalities of the fibrinogen molecule may lead to a compromised hemostatic function. We recently studied a 19 year old female who bled excessively following a child birth. She denied evidence of excessive bleeding or bruising in the past. A family history for a bleeding tendency was negative. Her prothrombin time was 14 seconds (control 12 sec); activated PTT was 26 seconds (control 25 sec); reptilase time was 34 seconds (control 13 sec); thrombin time was 44 seconds (control 19 sec); fibrinogen level was 79 mg/dl (control 250-350 mg/dl) by a clotting time method (Clauss's technique) and 384 mg/dl when measured immunologically. Similar abnormalities were found in her mother and three brothers. Studies related to the cleavage of her purified fibrinogen with thrombin, reptilase and venom from southern copperhead snake suggested an abnormality of her fibrinogen molecule, in that, the release of fibrinopeptide A from Aα chain was markedly impaired when compared with that of the purified normal fibrinogen. The immunological behavior of this fibrinogen was similar to that of normal on immunoelectrophoresis and two dimensional cross-electrophoresis. This first fibrinogen variant described from Virginia which appears to be inherited as dominant trait is designated as "Fibrinogen White Marsh" after the patient's place of origin in Virginia.

***Pseudomonas Putrefaciens* Bacteremia Associated with Chronic Venous Stasis Ulcers.** Mark Brantley, MD, and John Warner, MD, *Norfolk.*

Pseudomonas putrefaciens bacteremia is an extremely rare occurrence reported only twice in the English language literature. We have identified a third case and now propose a possible common relationship among these cases.

A 91-year-old woman with a 40 year history of chronic venous stasis and stasis ulcers presented with a one day history of fever and chills. Examination revealed a fever of 99°F, +++ edema on both lower extremities with an open stasis ulcer draining purulent material. The chronic changes on her lower extremities were characteristic of elephantiasis nostra verrucosa. Blood cultures on three separate occasions grew lactose non-fermenting, H_2S producing gram negative pleomorphic rods identified as *Pseudomonas putrefaciens*. The patient was treated with intravenous sulfamethoxazoletrimethoprim and recovered without incident.

Review of the previously reported cases, along with our case, demonstrates a common history of chronic venous stasis ulcers. Human isolates of *P. putrefaciens* have been found in a great variety of clinical specimens. However, the organism is generally considered a saprophyte in these settings.

Since *P. putrefaciens* is normally a non-invasive, psychrophilic organism. There must be determinants which have increased its virulence in these patients. We believe that the microbiologic environment of the devitalized tissue within the venous stasis ulcer provides a milieu suitable for increased virulence and thus systemic invasion. A lower temperature within the in vivo media of the venous stasis ulcer may be a key determinant.

Tick Transmitted Tularemia with Sporotrichoid Pattern. Barry Farr, MD, and W. Michael Scheld, MD, *Charlottesville.*

We report a case of sporotrichoid tularemia occurring after tick bite. Sporotrichoid tularemia has rarely been described and tularemia has declined in incidence nationally over the past 30 years. Virginia still ranks fifth, however, among states with most cases of tularemia per year. The majority of cases in Virginia, as in other eastern states occur in winter and involve hunters after contact with infected game such as rabbits. Fewer cases occur in summer and are mostly due to arthropod bites.

A 64-year-old farmer developed fever, headache and malaise three days after a tickbite in the right popliteal fossa in early June. A papule at the site of tick removal ulcerated over the next week and

lymphangitis progressed despite oral cephalexin for one week followed by parenteral penicillin, nafcillin and clindaymcin for five days. Five nontender subcutaneous nodules developed along the lymphangitic streak extending proximally from the ulcer, and femoral and inguinal adenopathy also developed. The sporotrichoid nodules and inguinal nodes became fluctuant. Aspiration yielded pus with many polymorphonuclear neutrophils but no bacteria on gram stain or culture. Tularemia agglutination titer was positive at 1:10,240. Streptomycin 20 mg/kg/day for nine days effected a cure.

Because most cases of suppurative lymphadenitis with lymphangitis are due to streptococci or staphylococci, tularemia is often not considered (as in this case for > two weeks). History of tick bite and negative gram stain of node aspirates should suggest this diagnosis. In addition, sporotrichoid tularemia, although rare, should be considered in the differential diagnosis of presumed sporotrichosis, especially if fever or constitutional symptoms are present.

Adult Respiratory Distress Syndrome (ARDS) as the Presenting Feature of Malignant Histiocytosis (MH). Thomas B. Gore, MD, Rebecca Romney, MD, David D. Stone, MD, and Charles E. Hess, MD, *Charlottesville*.

A 23-year-old man was hospitalized with marked pyrexia and hepatosplenomegaly. Within 48 hours there was rapid evolution of bilateral diffuse alveolar infiltrates with severe dyspnea, hypoxemia, and respiratory failure requiring mechanical ventilation. An open-lung biopsy demonstrated an inflammatory, desquamative alveolo-interstitial process consistent with ARDS, which was later shown to contain malignant histiocytes. Biopsy of a firm axillary lymph node revealed changes diagnostic of MH. There was no response to broad-spectrum antibiotics, and all cultures were negative. Combination chemotherapy was without effect and the patient died on the 60th hospital day of progressive respiratory failure. At autopsy, MH involved the lung, lymph nodes, liver, spleen, pleura, diaphragm, and central nervous system.

ARDS commonly occurs in association with shock, sepsis, or major trauma, none of which existed in our patient. While the exact pathogenesis of ARDS is unknown, we hypothesize that either secretory products of the malignant cells or the host's response to tumor invasion created a diffuse alveolar damage and fatal respiratory failure in our patient. This is the first reported case of ARDS due to malignant histocytosis.

Thrombocytopenia Resulting from Repletion of Iron in Iron Deficiency Anemia. Gerald A. Soff, MD, *Richmond*.

Thrombocytopenia is known to occur with iron deficiency, although normal or elevated platelet counts are more typical. In previously reported thrombocytopenic patients, iron repletion has resulted in an increase of platelet counts preceding or concomitant with reticulocytosis. A 17-year-old girl had severe chronic iron deficiency (Hgb 3.2, iron 22, transferrin 405) and an initially low normal platelet count (168,000) dropped following iron repletion (trough 21,000 on sixth day of therapy). Bone marrow aspirate revealed a marked decrease in megakaryocytes as well as myeloid and erythroid precursors and no stainable iron. The decrease in platelet counts approximated first order kinetics with half-life of 2.6 days and this slope is parallel to previous studies of platelet senescence. These data indicated that iron repletion resulted in severe, but temporary inhibition of platelet production. Reticulocyte and neutrophil counts began to rise within 48 hours of therapy, however, platelets began to rise only by day 9. The increase continued through day 18 of iron therapy (683,000). The mechanism of the decline in platelet counts is unknown. Folate and Vit. B12 levels were normal and the marrow was not megaloblastic. Iron is known to be both necessary for platelet production and yet limit excess production. In this case, an imbalance of the two iron dependent processes may have resulted in excess inhibition preceding the increase in platelet synthesis typically observed in thrombocytopenic, iron deficient patients after iron repletion is started.

Non-Menstrual Toxic Syndrome. R. J. Canterbury, MD, *Charlottesville*.

A 17-year-old white male was admitted to the hospital because of a 3-day history of lethargy, myalgias, headache, fever, rigors, rash and dizziness with standing. He had lanced a "boil" on his arm 5 days earlier. Physical exam revealed a lethargic white male with BP 100/60, P 60 and T 40.6°C. He had marked conjunctival injection and diffuse erythroderma with petecchiae on both feet. He had a 2.5 cm indurated lesion on his right arm and a 2 × 1 cm tender lymph node in his right axilla. He had a Gr I/VI SEM at the apex. Lab data included WBC 14,500 (60 B, 33 S, 7 L), HCT 44.9, TBil 1.7, LDH 453, SGOT 56, BUN 29, Creat 1.4, Phos 1.4, Ca 8.4 and CPK 2000. UA, CSF, CXR and ECG were normal. Blood, urine, throat, CSF and urethral cultures were negative. Aspirate of the arm lesion grew *S. aureus*.

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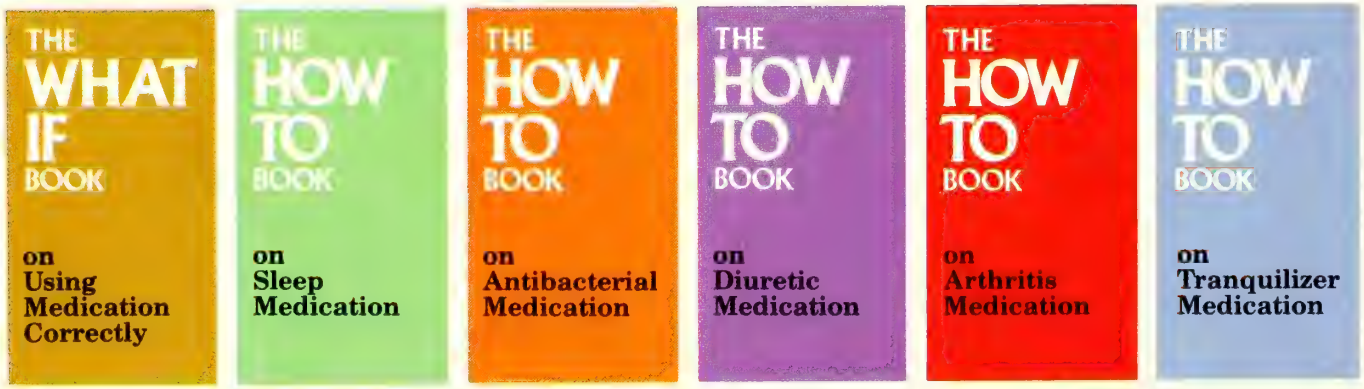
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He was treated with hydration, nafcillin and tetracycline. His symptoms improved significantly within 36 hours. Five days after admission he developed desquamation on his feet which generalized over the next week. Monospot, Weil-Felix, *Rickettsia* latex agglutination, tularemia and brucella titers and hepatitis screen were negative.

The case presented represents the toxic shock syndrome associated with staphylococcal infection. Although the typical patient seen with this syndrome is a menstruating female, we must be aware that it can be seen in any patient with a *S aureus* infection.

Infectious Mononucleosis Complicated by Fatal Aplastic Anemia. Michael T. Clayton, MD, Frederick Hayden, MD, Donald Innes, MD, and Charles Hess, MD, *Charlottesville*.

Epstein-Barr virus (EBV) infections have been associated with immunohematologic abnormalities ranging from transient granulocytopenia to neoplasia. Aplastic anemia is a rare but usually fatal complication of infectious mononucleosis (IM). We report an unusual case of heterophile-positive IM in a previously healthy 14-year-old boy complicated by fatal aplastic anemia and a fulminating lymphomatous meningoencephalomyelitis. The patient had a normal hematology profile and a negative monospot test three months before the onset of his present illness which began with fever, myalgias, pharyngitis, diffuse lymphadenopathy, hepatosplenomegaly and abdominal pain. An atypical lymphocytosis and a strongly positive monospot test confirmed the diagnosis of IM. Two weeks later, he developed severe pancytopenia progressing to bone marrow aplasia. Excepting three days of oral penicillin, he had no other drug, toxin or viral exposure. Administration of intravenous acyclovir (1500mg/M^2/day $\times$12d), methylprednisolone, antithymocyte globulin (20mg/kg/day $\times$8d) and a single infusion of high dose gamma globulin was without benefit. The patient developed multi-system failure, dominated by global central nervous dysfunction and CSF lymphocytic pleocytosis, and expired eight weeks after the onset of his symptoms.

Specific anti-EBV serologies were initially present although antemortem cultures of saliva, CSF and bone marrow were negative for EBV. At autopsy, the thymus, spleen and lymph nodes revealed lymphocyte depletion. Immunoperoxidase studies showed a diffuse, polyclonal lymphocytic infiltate of the CNS. We believe this patient represents an unusual and malignant case of IM, unresponsive to aggressive antiviral and immunoregulatory therapy.

Postviral Coronary Artery Aneurysm (Kawasaki's Disease) as a Cause of Myocardial Infarction? William Benton Lewis, MD, and Lockhart B. McGuire, MD, *Charlottesville*.

Unusual causes of coronary obstruction, especially in young persons, may include congenital coronary anomalies, trauma, hereditary connective tissue disorder, emboli, and a recently recognized viral arteriopathy producing arterial aneurysms (Kawasaki's disease).

A 20-year-old student was evaluated because of recent pulmonary congestion, cardiac enlargement, and an EKG pattern suggesting myocardial infarction. There was no background of chest pain, although there had been flu-like illnesses three weeks and two years earlier. There was no background of known "risk factors" for premature arteriosclerosis, and studies for abnormal immune complexes and active inflammation were negative. Cardiac catheterization confirmed a calcified, aneurysmal infarction of the anterior myocardium. The LAD artery was occluded by a recanalizing thrombus, probably in an arterial aneurysm, plus saccular dilatations in other coronary branches compatible with Kawasaki's disease. Treadmill test showed good overall cardiac reserve function; antithrombotic prophylaxis was started; and the patient has remained clinically well in the subsequent 18 months.

Kawasaki's disease (mucocutaneous node syndrome) has been described as an acute, febrile, exanthematous illness in children with cardiac involvement reported in 30% of subjects studied, and fatality in 1-2% because of myocardial infarction or aneurysm rupture. The specific infectious agent has not been identified. Other groups of young persons in the United States have been observed to have myocardial infarction after viral illness, presumably on the basis of viral arteriopathy.

Community Acquired *Legionella Micadei* Pneumonia. Thomas A. Alberico, MD, *Charlottesville*.

A 52-year-old white female with a diagnosis of recurrent diffuse histiocytic lymphoma (DHL) presented with a two-day history of fever, chills, pleuritic chest pain and nonproductive cough. Her DHL had been in complete remission since 1981. However, in July of 1982 recurrence was noted and on the day of admission retreatment with prednisone, procarbazine and cytoxan had just been completed. Pertinent clinical findings included an oral temperature of 40.1°C, left sided basilar rales and chest x-ray demonstrating left lower lobe infiltrate. Sputum gram stain was unremarkable. A white

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blood count of 2.2 k/mm^3 with 85% granulocytes was noted, with all other labs being normal. After 48 hours on Cefamandole, she had not defervesced, creatinine, BUN and liver enzymes were all elevated, and there was marked change in mental status (i.e. confusion, hallucinations). On the third hospital day open lung biopsy was performed with direct fluorescent antibody (DFA) of lung tissue revealing *Legionella micdadei* and antibiotic coverage was changed to erythromycin. Over the next three weeks of hospitalization all renal and hepatic functions as well as mental status normalized, however, all cultures remained negative for bacteria, AFB or fungi. Acute and convalescent *Legionella* titers are pending.

This case represents an excellent example of community acquired *Legionella micdadei*. The occasional manifestations of renal and hepatic insufficiency, and mental status changes (all previously described in *L. pneumophila*) were all present and eventually resolved. Although *L. micdadei* represents an infrequent pathogen outside the hospital, one must always be aware of its potential lethality and consider it in the differential diagnosis of the immune compromised host with pneumonia.

Can Left Main Coronary Artery Stenosis Be Detected by Quantitative Thallium-201 Scintigraphy? T. W. Nygaard, MD, R. S. Gibson, MD, J. A. Gascho, MD, G. A. Beller, MD, *Charlottesville*.

The utility of thallium (T1) scintigraphy for detection of patients with left main coronary artery (LMCA) disease has been questioned. Thus, quantitative exercise (Ex) (n = 37) or rest (n = 8) T1 scintigrams (scint) were analyzed in 45 pts. with LMCA stenosis (≥50%). Only 10 pts. (21%) demonstrated a "left main pattern" defined as ≥ 25% homogeneous decrease in T1 activity in the mid and upper septal and posterior walls on 45° LAO scint. However, 36 pts. (80%) had abnormal T1 uptake and/or washout in multiple vascular segments indicative of multivessel disease (MVD). Increased lung T1 was seen in 21/45 pts. (47%). All eight pts. imaged at rest due to unstable angina displayed patterns of LMCA disease (n = 4) or MVD (n = 4). Of 37 pts. undergoing Ex T1 scint, 26 (70%) had either ≥2.0 mm ST↓, Ex ↓BP ≥10 mmHG or ST↓ lasting ≥5 min post Ex as compared to 31/37 (84%) who had a high risk scint (LMCA, MVD or ↑ lung T1 patterns) (NS). Interestingly, 42/45 pts (93%) had either a markedly positive Ex stress test or high risk scint findings at Ex or rest. Only 1 pt (2%) had homogeneous initial T1 uptake and diffuse slow T1 washout.

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Reference: 1. Sheu YS, Ferguson JA, Cooper JR: *Evaluation of the Abuse Liability of Diethylpropion, Phendimetrazine, and Phentermine,* unclassified document, ADAMHA, HHS. Office of Medical and Professional Affairs, NIDA, 1980, pp 10–15.

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WARNINGS: Tolerance to the anorectic effect usually develops within a few weeks. When this occurs, the recommended dose should be discontinued. Phendimetrazine tartrate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly.
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ADVERSE REACTIONS: Cardiovascular: Palpitation, tachycardia, elevation of blood pressure.
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Endocrine: Impotence, changes in libido
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We conclude: 1) a typical scint pattern for LMCA disease is unusual (21%); 2) a markedly positive Ex stress test or a high risk scint is found in most (93%) pts. with ≥50% LMCA stenosis.

Use of Vasopressin in Esophageal Perforation. Alice Friedman, MD, *Salem*, and James Dill, MD, *Roanoke*.

Esophageal perforation is an uncommon, and frequently misdiagnosed cause of gastrointestinal bleeding. Delayed diagnosis results in increased mortality, with prompt surgical intervention reducing almost universal mortality to 30%. Esophageal perforation typically presents with the sequence of vomiting, minimal hematemesis, subxiphoid or substernal pain, dyspnea, and shock. Physical exam may be normal or reveal varying abdominal signs and chest findings. The chest radiograph frequently reveals effusions and/or pneumothorax, mediastinal widening, or subcutaneous emphysema. We present a patient who presented atypically with midepigastric pain and hematemesis, normal physical exam except for orthostatic changes, and normal chest and abdominal radiographs. Severe, persistent bleeding promptly ceased after initiation of systemic vasopressin. Early endoscopy revealed possible esophageal perforation, which was verified by gastrograffin studies and surgically repaired.

The use of systemic vasopressin has not been previously reported in cases of esophageal perforation. In this case, systemic vasopressin, in conjunction with early endoscopy, facilitated diagnosis of an emergent situation, directed choice of appropriate contrast studies, and provided advantageous preoperative stabilization. In cases of upper gastrointestinal bleeding with possible emetogenic esophageal injury, a high index of suspension, with prompt, aggressive therapy, may reduce the morbidity and mortality in this rare, but frequently fatal event.

Relapse of Lupus Transverse Myletis Mimicked by Vertebral Fractures and Spinal Cord Compression. Ann K. Henry, MD, and Carolyn M. Brunner, MD, *Charlottesville*.

We present a case of transverse myelitis associated with systemic lupus erythematosus (SLE) and the subsequent development of an unexpected non-disease-related complication mimicking transverse myelitis.

A 47-year-old housewife with SLE presented with acute onset of flaccid paralysis of the lower extremities. CSF exam showed no infection; spine films showed a partial collapse of T8; cervico-

thoracolumbar myelogram was negative. A diagnosis of anterior spinal artery syndrome was made and the patient improved with tapering doses of corticosteroids. Ten weeks later, however, she had a recurrence of symptoms and the weakness progressed despite an increase in prednisone. Spine films showed a posterior displacement of a compressed L1 vertebra and myelogram demonstrated a complete block at that level.

Neurological involvement with SLE has received increasing attention during recent years; transverse myelitis is one of the less frequently encountered syndromes, totaling only about 30 case reorts. This patient presented with just such a syndrome and when symptoms recurred, it was assumed that this was a relapse of the transverse myelitis. However, not all neurological changes in SLE patients are secondary to lupus activity. High dose corticosteroid therapy was presumably responsible for the compression fractures and eventual progressive neurological deterioration in this patient.

Successful Therapy of Sporotrichosis (S) with Ketoconazole (K) with Endocrine Complications. Ronald M. Ovetsky, MD, and Charles J. Schleupner, MD, *Salem.*

Sporotrichosis, an occupational infection of horticulturists, usually presents as lymphocutaneous disease and responds to potassium iodide. K is a new broad spectrum oral antifungal agent which blocks fungal sterol synthesis. Variable results have been reported in the seven patients with S previously treated with K. We report a 33-year-old male with a six-week history of a right index finger lesion with associated nodules in the arm, axilla and right neck. Therapy with K (400 mg/day) was initiated and the diagnosis was confirmed by culture. The patient responded with complete healing. Therapy was accompanied by nausea and diminished libido. While gynecomastia and depressed serum levels of testosterone (T) and cortisol (C) have been reported in a few patients, clinically evident sexual dysfunction (and hypoadrenalcorticalism) have not been previously reported. Serum T and C levels obtained before and four days after initiation of K are shown:

	Pre-therapy	4 hours after dose
Serum T (ng/dl)	464	97
Plasma C (μg/dl)	17.7	2.0

Additionally, an ACTH stimulation test, as well as LH, FSH, 17-OH progesterone, desoxycortisol and free and total T levels were obtained during therapy. The results of these studies will be presented. While K is an effective antifungal, its side effects may limit its usefulness for needed long-term therapy.

Primary Hypothyroidism and Hyperprolactinemia in an Ateliotic Dwarf. Robert F. Davis, MD, *Norfolk.*

A case is presented of a short statured young woman (ateliotic dwarf) with mastodynia and evidence of isolated growth hormone deficiency. Extensive testing revealed normal thyrotropin, gonadotropin, and corticotropin function with growth hormone unresponsive to insulin stimulation. The patient was found to have a goiter with borderline low T_3, T_4, T_7, but an elevated TSH (42 micro I.U.) which rose further after TRF. In addition, antimicrosomal and antithyroglobulin antibodies were elevated (1:25,000 and 1:1600 respectively). Prolactin levels were also elevated (55 ng/ml). TSH but not prolactin levels returned to normal following thyroid replacement. A syndrome of ateliotic dwarfism, goiter, and mastodynia associated with hyperprolactinemia and lymphocytic thyroiditis is presented. The occurrence of these rare conditions together suggests a common etiology.

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VIRGINIA MEDICAL

EDITORIAL

Fitness Mania: A Threat to Private Practice

Not long ago, I had an interesting chat with an internist friend at a social gathering. He had received a brochure for a sports medicine fitness conference of which I was course chairman. Since he was not an orthopedic surgeon or physical therapist, he was having great difficulty understanding why he was included on the mailing list for this conference.

Now, my internist friend, whose subspecialty is diabetes, is about 50 years of age, 30 pounds overweight, and not athletically inclined. In my travels around the country, I have observed that he has plenty of company. The stereotype characteristics also include no formal medical education concerning fitness, well-being, exercise, or nutrition. He is content to look at the practice of medicine as crisis management resolved mainly by reaching into his pharmacological "goody bag." In short, his definition of good health is *absence from disease*!

Whether we like it or not, the public in increasing numbers is defining good health as "maximum physiological attainment." Whether we like it or not, the public is starting a fitness binge, and the bottom-line facts are that the majority of physicians are unaware of the onset. Like my internist friend, they do not perceive either the magnitude or the importance of the movement.

The importance of the situation is twofold. First, the quality of medical care can clearly be enhanced by better appreciation and implementation of fitness programs. Second, dramatic changes are taking place in the economics of medical care.

There are hazards and pitfalls, to be sure. Injuries occur, mainly because the public has been given little in proper guidelines. I believe physicians as a whole have abdicated their responsibility to the public through disinterest, ignorance, or both. Is it any wonder that excesses and injuries occur when the only supervision and education comes from TV show hosts, actresses, dancers, and 20-year-old weight-lifting instructors at the local health spa? Where is the medical community, when life-threatening diet fads become best sellers?

In spite of the excess, the hype, and the injuries, it is quite clear that well-designed fitness programs can help—and, indeed, help a lot! Where is the public to get well-designed programs? Should my internist friend be able to offer a first-class program for weight loss and fitness to his deconditioned diabetic patients? You bet! Yet he, like thousands of other physicians, has yet to appreciate the importance of this situation. If delivering better health care is not motivation enough, perhaps health care economics will be.

Physician apathy, disinterest, and ignorance may be commonplace, but the public is moving forward. Thousands of aerobics dance classes, health spa chains, racquet ball and jogging clubs, and what-have-you have sprung up across the nation. Nike,

Photograph courtesy Richmond Newspapers, Inc.

the jogging shoe firm, had gross sales of $8,000 in 1964; in 1981, its annual gross sales were $450 million! The public is voting with its pocketbook. Believe, me, the fitness-health-care dollar is moving to gyms, sports clubs, weight reduction chains, dance classes, you name it. Pritikin Diet Centers proclaim freedom from hypertension, coronary occlusion, and diabetes. Some 80 million Americans are on the fitness march. If physicians do not take heed, they may be wondering where all the patients went.

If you, as a physician, do not define good health as maximum physicological capacity and help your patients attain it, they simply will go elsewhere, taking their dollars with them.

The next decade will be extremely interesting. In my view, the fitness boom of the '80s can be a much greater threat to the private practice of medicine than the governmental threats of the '70s. It's up to you. The public's needs and wants are changing. Are you prepared for what's coming?

ROBERT P. NIRSCHL, MD

Hallmark of a Long Practice: Quality

A FEW weeks ago the Medical Staff of Hampton General Hospital gathered briefly to honor one of its members. The occasion was his retirement after 55 years of practice. In deference to the honoree's quiet ways, the celebration was subdued. The man and the practice honored at that event, however, are truly exceptional.

Dr. Eldred S. Jones was born and raised in Hampton. After an outstanding academic career at the University of Virginia, he returned in 1927 to practice medicine and surgery in his community. It would be impossible to count the patients Dr. Jones has served these many years, but it is not the length or volume of his practice that is so remarkable. Rather, it is the quality of that practice that is so impressive.

Two things stand out. The first is his unfaltering patience. No one can ever remember Dr. Jones being hurtful or short-tempered. Anyone in the healing trade knows some sick humans can be prickly. Dr. Jones has always been able to absorb these barbs and react as we all should, with kindness and understanding. As witness to this, in the 18 years I have worked in Hampton, I have never met a person who was angry with Dr. Jones.

The other remarkable quality of Dr. Jones' practice is his devotion to academic discipline. About a month before he decided to retire, an old patient returned just to refresh a prescription for antispasmodics. The man had a long-standing functional GI problem and many of us would have been content to rewrite the prescription and send the patient on his way. Because Dr. Jones had not seen this man for a couple of years, and true to the rules of good medical practice, Dr. Jones examined the offending abdomen. He immediately recognized a newly developed aortic aneurysm which was subsequently shown to be 11 centimeters in diameter and was successfully resected.

A medical practice that covers half a century, that maintains academic quality throughout, and that leaves all the customers as friends sets a standard to which we may all aspire.

JESS P. MILLER, MD

2019 Cunningham Drive
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Editor's Note: Dr. Jones died on January 11 in Hampton at the age of 82.

VIRGINIA MEDICAL OBITUARY

Emerson M. Babb, MD

Dr. Emerson McCaulay Babb, Ivor family practitioner and internist for 44 years, died in a Richmond hospital on December 18, 1982, following a long illness. He was 85 years old.

An Ivor native, Dr. Babb was graduated from the Medical College of Virginia, interned at Richmond's Stuart Circle Hospital, and returned to his hometown to practice. He was a veteran of both world wars and was commander of a hospital train in Europe during World War II. Following the war and postgraduate training in internal medicine in Chicago and New York, he resumed his practice in Ivor, serving on the staff of Louise-Obici Memorial Hospital in Suffolk. On his retirement in 1968, he moved to Richmond.

A member of the Southern Medical Association and American Medical Association, Dr. Babb was a past president of the Tri-County Medical Society. His membership in The Medical Society of Virginia spanned 57 years. A son, Dr. John R. Babb is an ophthalmologist practicing in Beaumont, Texas.

I. Floyd Nesbitt, MD

Dr. Isaac Floyd Nesbitt, long time Newport News family physician, died October 27, 1982, at the age of 62.

A native of Hendersonville, North Carolina, Dr. Nesbitt completed undergraduate studies and the first two years of medical school at the University of North Carolina, Chapel Hill. He then transferred to the University of Maryland, Baltimore, and received his medical degree in 1943. Following internship at the Medical College of Virginia and residency in obstetrics and gynecology at Riverside Hospital, Newport News, Dr. Nesbitt joined the service and, at the end of World War II, was stationed in Japan. He returned in 1947 to establish a family practice in Newport News. Dr. Nesbitt joined the staff of the Hampton Veterans Administration Center in 1969, and served there until his death.

For 35 years a member of The Medical Society of Virginia, Dr. Nesbitt was also a member of the Newport News Medical Society.

Memoir of W. H. Chapman 1904–1982

By R. W. Garnett, MD

Dr. William Hardee Chapman died in Charlottesville on August 26, 1982, after a prolonged illness at the age of 78.

Dr. Chapman was born in Chester, South Carolina, and received his MD from the University of Maryland in 1929. He took internship at Women's Hospital in Baltimore, Maryland, and residency in medicine and surgery at Petersburg, Virginia, General Hospital. He was a medical officer in the Veterans Administration from 1932 to 1940 when, after losing his sight in both eyes, he took training in psychiatry at the University of Cincinnati and at Vanderbilt, becoming a specialist in the use of hypnosis in psychotherapy.

In 1953 he moved to Charlottesville, where his first wife took a position on the faculty of the University of Virginia School of Nursing and he became a clinical instructor and later clinical assistant professor in the Department of Psychiatry, working in the Outpatient Clinic.

Bill Chapman was a genial figure around the university. He was a faithful member of the Albemarle County Medical Society, attending every meeting he was able to and relishing the fellowship. He turned his misfortune to good use and was never bitter or defeated. As a hobby, he became an accomplished ham radio operator and cultivated friends through this medium all over the country. He suffered more than his share of illness and misfortune but remained cheerful and uncomplaining.

We honor his memory.

Prepared and published at the request of the Albemarle County Medical Society.

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There are many excellent handbooks of effective writing, among them *The Elements of Style,* by William Strunk, Jr., and E. B. White (MacMillan); *The Careful Writer: A Modern Guide to English Usage,* by Theodore M. Bernstein (Atheneum); and *How to Write and Publish a Scientific Paper,* by Robert A. Day (ISI Press).

WHO'S WHO

To **Dr. Paul C. Pearson**, Warsaw, went The Medical Society of Virginia's highest honor, the Physician Award for Community Service, during ceremonies at the annual meeting in Williamsburg. **Dr. H. C. Alexander III**, MSV President, presented the award.

A general practitioner who earned his doctorate in medicine in 1933 at the Medical College of Virginia, Dr. Pearson first practiced briefly in Aylett, then moved to Warsaw, where he has practiced for the past 35 years.

In Warsaw he became a charter member of the King William County Ruritan Club, was elected to the Town Council, and joined the Warsaw Rotary Club and the Warsaw Baptist Church. He serves on the board of trustees of the Mattaponi Indian Reservation and is a lifetime member of many medical organizations and a fellow of the Royal Society of Health.

To all these groups, Dr. Pearson has contributed with "great enthusiasm and energy," said Dr. Alexander, who concluded the citation with this characterization of the recipient: "A sympathetic friend, wise counselor and beloved physician who has dedicated his life and his efforts to serving his community."

Dr. Pearson is the 20th Medical Society of Virginia member to receive the award, which was established in the early 1960s by the A. H. Robins Company and has continued ever since under the firm's sponsorship.

An exhibit entered by **Dr. George W. Vetrovec**, Richmond, took first prize in the scientific exhibit area at The Medical Society of Virginia's annual meeting in Williamsburg. Coexhibitors were **Dr. Michael J. Cowley** and **Dr. Andrea Hastillo**, also of Richmond, and the title of the display was "Intracoronary Thrombolysis in Acute Myocardial Infarction: Technique and Results."

Second place went to the exhibit on iatrogenic renal disease by **Dr. Benjamin C. Sturgill**, Charlottesville, and third prize was won by **Dr. Darrell K. Gilliam**, **Dr. Epes Harris** and **Dr. Maurice Wood**, all of Richmond, for their exhibit showing some results of nadolol/hydrochlorothiazide therapy in hypertensive patients.

Three Medical Society of Virginia members were recently named fellows in the American Academy of Family Physicians. They are **Dr. J. Thomas Ryan**, Fredericksburg, **Dr. Robert A. W. Latimer**, Manassas, and **Dr. G. F. Sutherland**, Big Stone Gap.

The American College of Physicians has conferred fellowship on these MSV members: **Dr. John H. Armstrong**, Winchester; **Dr. Stuart B. Baker**, Norfolk; **Dr. Carl J. Friedman**, Crozet; **Dr. Ronald J. Karpick**, Falls Church; **Dr. Thomas J. O'Neill**, Danville; **Dr. Thomas E. Patteson III**, Front Royal; and **Dr. Shaun J. Ruddy**, Richmond.

The American College of Surgeons has named to fellowship **Dr. C. Thomas Harvey**, Danville.

Super civics: Dr. Paul C. Pearson, Warsaw (left) holds the Physician Award for Community Service. With him is longtime friend Dr. Norman R. Tingle.

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Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

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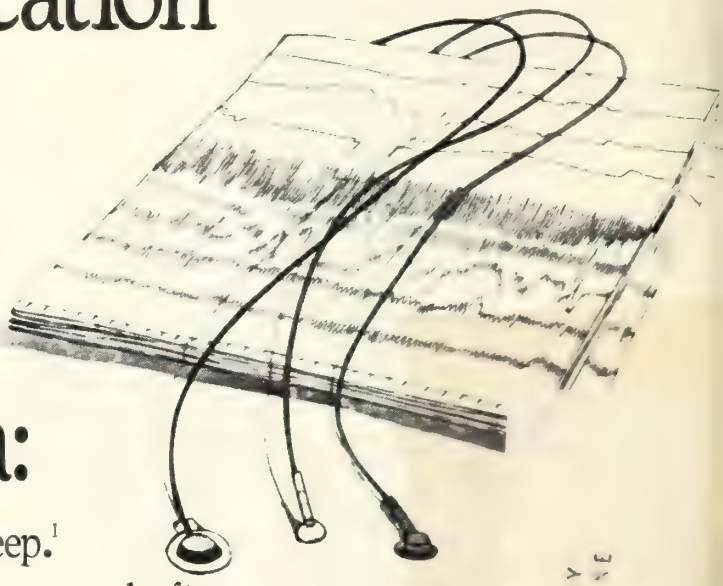
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Volume 110 March 1983 Number 3

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March 1983, Volume 110, Number 3

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ACTIONS OF COUNCIL

At its meeting on January 22, the Council:

Thank-you note **Received** from Dr. Raymond S. Brown a framed color photograph of Walter Reed's birthplace, which The Medical Society of Virginia's gifts of money help to maintain.

Political action **Heard** Dr. Percy Wootton urge increased support for VaMPAC this year, when 140 of Virginia's delegates and senators will be elected. (For pictures of a VaMPAC salute to the General Assembly leadership, see pages 138-141.)

More political action **Listened** to the Society's counsel, Allen C. Goolsby III, suggest that the dinners for legislators hosted annually by councilors in their districts be changed to breakfasts or receptions or otherwise given a fresh approach. Attorney Goolsby also described medical legislation in the hopper at the 1983 General Assembly and forecast that the cap on professional liability awards would be increased from $750,000 to $1 million.

Life insurance **Approved** a new life insurance program, jointly recommended by the Membership and Insurance Committees, that would furnish $10,000 in term life insurance to new members. Cost to the Society: $2 per member for the initial year, after which the policyholders would be billed at the current premium. Dr. J. Thomas Hulvey, Membership Committee chairman, brought the recommendation to Council, pointing out that the program would be an inducement in the solicitation of new members.

CME results **Applauded** the report of Dr. Kinloch Nelson, Director of Continuing Education, that only 20 physicians have been lost to membership due to failure to comply with the CME program initiated in 1979. Total current MSV membership: 6,232.

Liability series **Learned** that the series of ten risk management seminars sponsored jointly over the state by the St. Paul Insurance Companies and The Medical Society of Virginia have drawn a total of 1,254 physicians representing virtually all specialties. This CME activity is under Dr. Nelson's wing, and he reported the attendance figure.

Impaired MD brochure **Learned** that a brochure is being worked up to describe the new Physicians' Health and Effectiveness Program, with the view to sending it to all physicians licensed in Virginia. The leaflet will tell how the program operates and how to report. It was noted that legislation had been introduced at the General Assembly to provide immunity for members of the program's committee, intervenors, and investigators.

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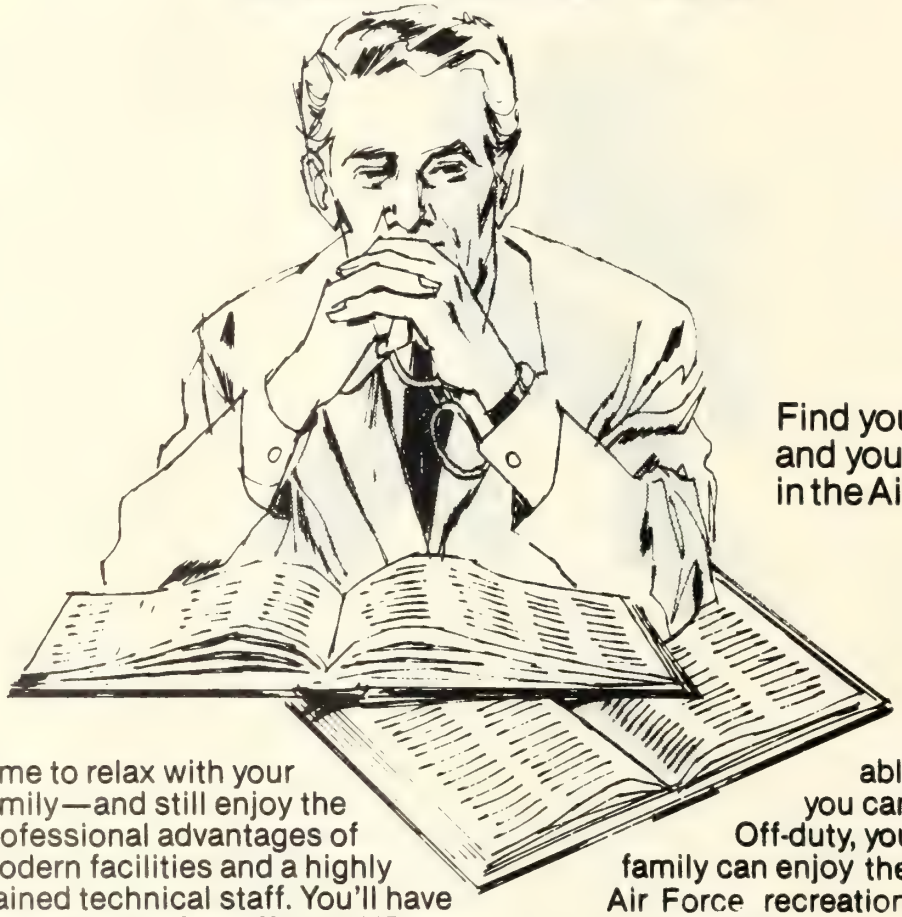

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Reference: 1. Sheu YS, Ferguson JA, Cooper JR: *Evaluation of the Abuse Liability of Diethylpropion, Phendimetrazine, and Phentermine,* unclassified document, ADAMHA, HHS. Office of Medical and Professional Affairs, NIDA, 1980, pp 10-15.

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WARNINGS: Tolerance to the anorectic effect usually develops within a few weeks. When this occurs, the recommended dose should be discontinued. Phendimetrazine tartrate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly.

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USAGE IN PREGNANCY: The safety of phendimetrazine tartrate in pregnancy and lactation has not been established. Therefore, phendimetrazine tartrate should not be taken by women who are or may become pregnant.

USAGE IN CHILDREN: Phendimetrazine tartrate is not recommended for use in children under 12 years of age.

PRECAUTION: Caution is to be exercised in prescribing phendimetrazine tartrate for patients with even mild hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of phendimetrazine tartrate and the concomitant dietary regimen. Phendimetrazine tartrate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage.

ADVERSE REACTIONS: Cardiovascular: Palpitation, tachycardia, elevation of blood pressure.
Central Nervous System: Overstimulation, restlessness, dizziness, insomnia, euphoria, dysphoria, tremor, headache; rarely psychotic episodes at recommended doses.
Gastrointestinal: Dryness of the mouth, unpleasant taste, diarrhea, constipation, other gastrointestinal disturbances.
Allergic: Urticaria
Endocrine: Impotence, changes in libido.

OVERDOSAGE: Manifestations of acute overdosage with phendimetrazine tartrate include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states.
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Political activists salute top lawmakers

Big givers to VaMPAC gave a big party for leaders of Virginia's legislature on the eve of the 1983 General Assembly's opening session. The hosts were PAC 250 members. The honorees were the legislators who wield gavels, head up caucuses, manage majorities and minorities, and chair committees. The setting was the ballroom of the Commonwealth Club in downtown Richmond.

There was a bar, and several buffets, and candlelit tables, and a great ice sculpture of the Capitol. There were lots of legislators and lots of physicians, many more than the photographs on these four pages encompass.

On the PAC side there were leaders, too, including four former Medical Society of Virginia presidents: Dr. Percy Wootton, who as VaMPAC chairman greeted guests at the door; Dr. W. Leonard Weyl, board member of the American Medical Political Action Committee; Dr. William S. Hotchkiss, AMA trustee and secretary-treasurer; and Dr. Raymond S. Brown.

Finally, there was Gov. Charles S. Robb himself, stopping by with Mrs. Robb to put his seal of approval on the evening. A.G.

▲ **Sen. Stanley C. Walker (D-Norfolk), Laws Committee chairman, left, and Dr. Percy Wootton.**

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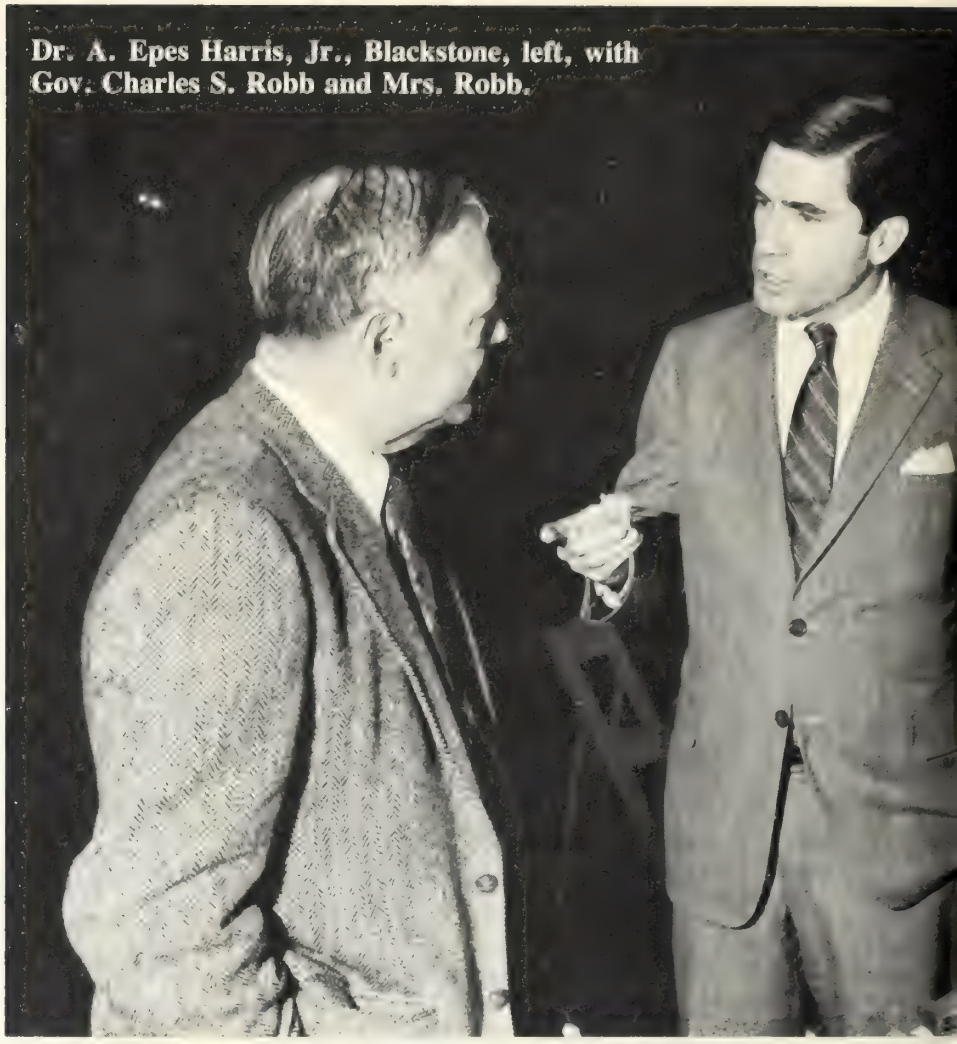
▼ Dr. Gervas S. Taylor, Jr,, Norfolk, left, and Speaker of the House A. L. Philpott (D-Bassett).

All photographs by Tim Wright

▲ Del. Owen B. Pickett (D-Virginia Beach) of the Health Professions Committee, left, and Dr. George E. Broman, Jr., Culpeper.

▲ Dr. William S. Hotchkiss, Chesapeake, left, and Del. George W. Jones, Bon Air, Republican caucus leader.

▼ Dr. Antonio M. Longo, Alexandria (left), Dr. H. Michael Hanna, Jr., Fort Defiance (center), and Del. Alson H. Smith, Jr., Winchester, Democrat caucus leader.



Dr. A. Epes Harris, Jr., Blackstone, left, with Gov. Charles S. Robb and Mrs. Robb.

▲ Del. Thomas W. Moss, Jr. (D-Norfolk), House majority leader, left, and Dr. Joe W. King, Richmond.

▲ Del. Vincent F. Callahan (R-McLean), House minority leader, left, and Dr. W. Leonard Weyl, Arlington.

▲ Del. Mary A. Marshall (D-Arlington), of the Health and Aging Committee, and Dr. C. Barrie Cook, Fairfax.

▲ Dr. Bedford C. Berrey, Richmond, left; Attorney General Gerald Baliles, center; and Dr. J. William Giesen, Radford.

▲ Dr. Harold I. Nemuth, Richmond, left, and Sen. W. F. Parkerson, Jr. (D-Richmond), Courts of Justice chairman.

▲ Dr. Wendell J. Pile, Newport News, left, and Del. Richard M. Bagley (D-Hampton), of the Appropriations Committee.

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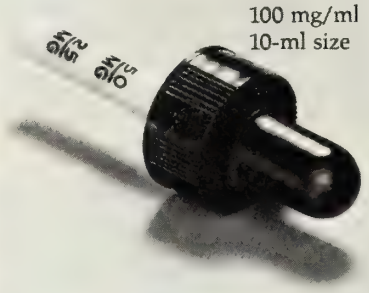

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Wise County Medical Society

Abul Hashem, MD, Radiology, PO Box 1800, Wise VA 24293

BRIEF SUMMARY
PROCARDIA® CAPSULES For Oral Use
(nifedipine)

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request

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Pfizer LABORATORIES DIVISION
PFIZER INC

"I can do things that I couldn't do for 3 yrs. including joining the human race again."

Quotes from an unsolicited letter received by Pfizer from an angina patient. While this patient's experience is representative of many unsolicited comments received, not all patients will respond to Procardia nor will they all respond to the same degree.

© 1983, Pfizer Inc.

"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA[*] *as soon as it became available. The change in my condition is remarkable."*

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks, taking fewer nitroglycerin tablets, doing more, and being more productive once again

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).

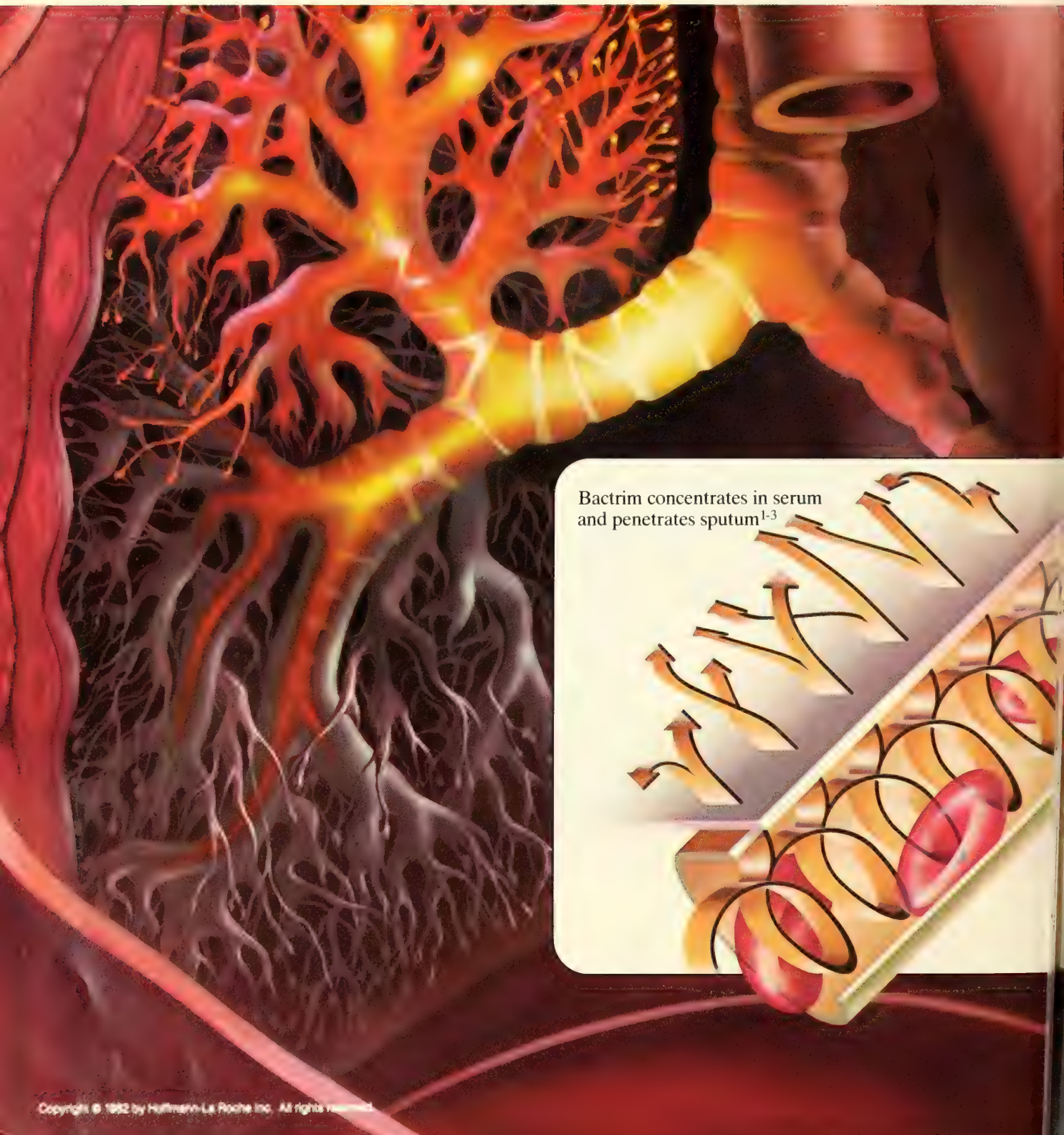
for the varied faces of angina

PROCARDIA®
(NIFEDIPINE) *Capsules 10 mg*

*Procardia is indicated for the management of:
1) Confirmed vasospastic angina.
2) Angina where the clinical presentation suggests a possible vasospastic component
3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

Please see PROCARDIA *brief summary on adjoining page*

Bactrim™ attacks the
(trimethoprim and sulfamethoxazole/Roche)
in acute exacerbations



Bactrim concentrates in serum and penetrates sputum[1-3]

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ROCHE

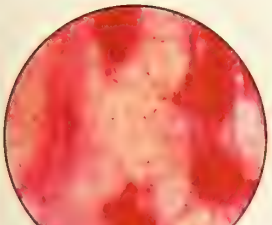
major pathogens of chronic bronchitis*

Bactrim clears sputum of susceptible bacteria

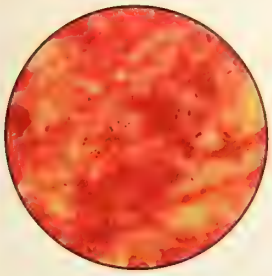
In sputum cultures from patients with acute exacerbations of chronic bronchitis, *H. influenzae* and *S. pneumoniae* are isolated more often than any other pathogens.[4,5] One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens.[5]

Bactrim is effective *in vitro* against most strains of both *S. pneumoniae* and *H. influenzae*—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.[6]

attacks *H. influenzae*—even ampicillin-resistant strains



attacks *S. pneumoniae*



Bactrim reduces coughing and sputum production

In three double-blind comparisons with ampicillin *q.i.d.*, Bactrim DS proved equally effective on all clinical parameters.[7-9] Bactrim reduced the frequency and severity of coughing, reduced the amount of sputum produced and cleared the sputum of purulence.

Bactrim has the added advantages of *b.i.d.* dosage convenience and a lower incidence of diarrhea than with ampicillin, and it is useful in patients allergic to penicillins.

Bactrim also proved more effective than tetracyclines in 10 clinical trials involving nearly 700 patients.[10] Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

References: 1. Hughes DTD, Bye A, Hodder P: *Adv Antimicrob Antineoplastic Chemother 1/2*:1105-1106, 1971. **2.** Jordan GW *et al: Can Med Assoc J 112*:91S-95S, Jun 14, 1975. **3.** Beck H, Pechere JC: *Prog Antimicrob Anticancer Chemother 1*:663-667, 1969. **4.** Quintiliani R: Microbiological and therapeutic considerations in exacerbations of chronic bronchitis, in *Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts;* Princeton Junction, NJ, Communications Media for Education, Inc., 1980, pp. 9-12. **5.** Schreiner A *et al: Infection 6*(2):54-56, 1978. **6.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **7.** Chodosh S: Treatment of acute exacerbations of chronic bronchitis: results of a double-blind crossover clinical trial, in *Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts. Op. cit.*, pp. 15-16. **8.** Chervinsky P: Double-blind clinical comparisons between trimethoprim-sulfamethoxazole (Bactrim™) and ampicillin in the treatment of bronchitic exacerbations. *Ibid.*, pp. 17-18. **9.** Dulfano MJ: Trimethoprim-sulfamethoxazole vs. ampicillin in the treatment of exacerbations of chronic bronchitis. *Ibid.*, pp. 19-20. **10.** Medici TC: Trimethoprim-sulfamethoxazole (Bactrim™) in treating acute exacerbations of chronic bronchitis: summary of European clinical experience *Ibid.*, pp. 13-14.

Economical b.i.d.

Bactrim™ DS

(160 mg trimethoprim and 800 mg sulfamethoxazole/Roche)

*Due to susceptible organisms. **Please see next page for summary of product information.**

Bactrim™

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides, patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General.* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy. Teratogenic Effects. Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias.* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions.* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions.* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions.* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions.* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN

Adults. Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children. Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment. Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS

Usual adult dosage. 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100, Tel-E-Dose® packages of 100, Prescription Paks of 20 and 28. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500, Tel-E-Dose® packages of 100, Prescription Paks of 40. *Pediatric Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml), cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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A Blueprint for a Community's Health: The New Stuart Circle Medical Building

Charter Medical Corporation proudly announces the opening of its Stuart Circle Medical Building on Richmond's historic Monument Avenue.

The new medical building offers physicians the opportunity to become an integral part of an active community. Located in the center of the beautiful Fan district, the newly renovated office building provides space for physicians to practice, either solo or in groups.

The office is within half a block of Stuart Circle Hospital, a 153-bed, short-term, medical/surgical hospital offering the convenience of outpatient testing for your patients. A 24-hour Outpatient Department and many other ancillary services are also available at the hospital.

Although renovated for modern-day patient/physician comfort, much of the townhouse-style flavor remains, including fireplaces and mantels in selected offices. The floor-plan design insures convenience and patient privacy in both the consultation and examination areas. The decor matches the traditional elegance of the surrounding historical homes.

Applications are now being accepted for office space. Interested physicians should call project manager Ray Hayes at (804) 359-1843.

The Stuart Circle Medical Building
1617 Monument Avenue
Richmond, Virginia

—A CHARTER MEDICAL CORPORATION FACILITY—

MEETINGS

March 25-27
Audiovisual Symposium on Short Term Dynamic Psychotherapy (Virginia Institute of Short Term Dynamic Psychotherapy), *Charlottesville*. 21 Cat. I credits. Fee: $150. Jason Worchel, MD, 1932 Arlington Blvd., Charlottesville VA 22903, (804) 977-6644.

March 27
Sports Medicine for Women (Eastern Virginia Medical School), *Norfolk*. Lou Ann Hamer, EVMS, PO Box 1980, Norfolk VA 23501.

April 6
Annual Clinical Conference (Louise-Obici Memorial Hospital), *Suffolk*. Cat. I credits. PO Box 1100, Suffolk VA 23434, (703) 539-1511, ext. 2692.

April 7-8
Ninth Annual Symposium on Diagnosis and Treatment of Neoplastic Diseases (Johns Hopkins University), *Baltimore*. Diane Hexdinger, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-6046.

April 8-9
Clinical Gastroenterology for Primary Care Physicians (University of Maryland), *Baltimore*. Office of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

April 8-10
Acute Trauma, Diagnosis and Management (Medical College of Virginia), *Williamsburg*. Fee: $250. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

April 11-16
Pediatric Trends (Johns Hopkins University), *Baltimore*. 45 Cat. I credits. Edward M. Sills, MD, Johns Hopkins Hospital, Baltimore MD 21205.

April 13-15
First Annual Southeast Regional Gerontology Conference (Eastern Tennessee Baptist Hospital), *Knoxville, Tennessee*. Sheila Gordon, PO Box 1788, Knoxville TN 37901, (615) 632-5061/5707.

April 14
Innovations in Lung Cancer (American Cancer Society, Northern Virginia Area), *Arlington*. Cat. American Cancer Society, Northern Virginia Area, (703) 938-5550.

April 15-16
Symposium on Non-Invasive Cardiac Imaging (Virginia Heart Institute), *Richmond*. Karen Wormley at the Institute, 205 N. Hamilton St., Richmond VA 23221, (804) 359-9265.

April 15-17
Clinical Concerns in Primary Care (Medical College of Virginia) *Williamsburg*. Fee: $195. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

April 17-20
Current Topics in Allergy and Clinical Immunology (American Academy of Allergy and Immunology/University of Michigan), *Williamsburg*. 10½ Cat. I credits. Fee: $225. Office of CME, Towsley Center, University of Michigan Medical School, Ann Arbor MI 48109, (313) 763-1400.

April 17-22
Operative Treatment of Fractures and Non-Unions (Johns Hopkins University), *Hot Springs*. 36 Cat. I credits. Fee: $685. Carlita Kearney, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-3168.

April 18-22
Annual Meeting, American Roentgen Ray Society, *Atlanta*. R. A. Gagliardi, MD, 880 Woodward Ave., Pontiac MI 48053.

April 19
Annual Charles W. Thomas Lecture on Rheumatology: Pathogenesis of Rheumatoid Arthritis (Medical College of Virginia), George Ben Johnston Auditorium, *Richmond*. Shaun Ruddy, MD, Box 263, MCV Station, Richmond VA 23298.

April 21
14th Annual Symposium on Medicine and Religion: "The Impaired Professional" (Portsmouth Naval Hospital and The Medical Society of Virginia), Auditorium, Main Hospital Building, Naval Regional Medical Center, *Portsmouth*. Capt. G. E. Paulson, MD, (804) 398-5559.

April 21-23
Diagnosis and Treatment of Common Human Tumors (American Cancer Society, Kentucky Division), *Lexington, Kentucky*. American Cancer Society, Kentucky Division, 1169 Eastern Parkway, Louisville KY 40217.

April 21-23
Sports Medicine and Fitness Symposium (Georgetown University), *Washington DC*. Cat. I credits. Robert P. Nirschl, MD, 3801 Fairfax Dr., Arlington VA 22203, (703) 525-8000/2000.

April 22-24
Arrhythmia and Cardiac Ischemia: Diagnosis and Management (International Medical Education Corporation), *Williamsburg*. Cat. I credits. Mary A. Schroeder, IMEC, 64 Inverness Dr., E., Englewood CO 80112, toll free: (800) 525-8651.

continued over

April 22
4th Annual Spring Clinical Conference (Medical College of Virginia and John Randolph Hospital), Holiday Inn, *Chester*. Theodore C. Andrew, MD, John Randolph Hospital, Hopewell VA 23860.

April 23
Biomedical Consequences of Nuclear Weapons and Nuclear War (University of North Carolina), *Chapel Hill*. 7 Cat. I credits. Betty Neilson, Office of CME, 231 MacNider Bldg., Chapel Hill NC 27514, (919) 962-2118.

April 28-30
Sexually Transmissible Diseases: An Update (Johns Hopkins University), *Baltimore*. 18 credit hrs. Fee: $250. Susan Bavaro, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

April 28-30
Emergency Medicine (Medical College of Virginia), *Williamsburg*. Fee: $195. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

April 28-30
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Pain Symposium (University of Maryland), *Baltimore*. Office of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

April 29-May 1
Clinical Management of Coronary Disease and Dual Mode Management (International Medical Education Corporation), *Arlington*. Cat. I credits. Mary A. Schrorder, IMEC, 64 Inverness Dr., E., Englewood CO 80112, toll free: (800) 525-8651.

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Annual Meeting, American Federation for Clinical Research, *Washington D C*. C.B. Slack, 6900 Grove Rd., Thorofare NJ 08086.

April 30
1st Annual Spring Symposium (Eastern Virginia Interhospital Medical Education Committee), *Kilmarnock*. Robert Hoyt, MD, Rappahannock General Hospital, Kilmarnock VA 22482.

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Annual Meeting, Virginia Society of Ophthalmology and Otolaryngology, *Charlottesville*. Donna Strawderman, 4205 Dover Rd., Richmond VA 23221, (804) 353-2721.

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Current Topics in Clinical Infectious Diseases (University of Virginia), *Roanoke*. 6 Cat. I credits. Fee, $30. Charles J. Schleupner, MD, (703) 982-2463.

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Annual Meeting, American Geriatrics Society, *New York, New York*. K. Henderson, 10 Columbus Circle, New York NY 10019.

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Topics in Cardiovascular Disease (American Heart Association/Irish Cardiac Society), *Baltimore*. Michaeline Silverstein, PO Box 17025, Baltimore MD 21203, (301) 685-7074.

May 16-18
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Clinical Radiation Oncology (Medical College of Virginia/Virginia Commonwealth University), *Williamsburg*. Fee: $250. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

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National Conference: Breast Cancer 1983 (American Cancer Society), *Boston, Massachusetts*. 16½ credit hrs. Nicholas G. Bottiglieri, MD, American Cancer Society, 777 Third Ave., New York NY, 10017, (212) 371-2900.

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May 28-30
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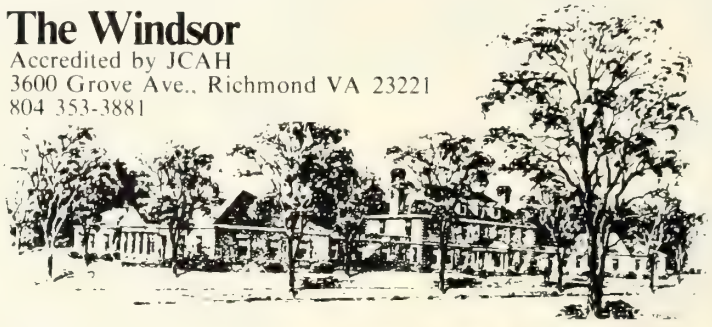

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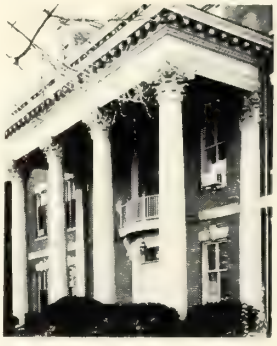

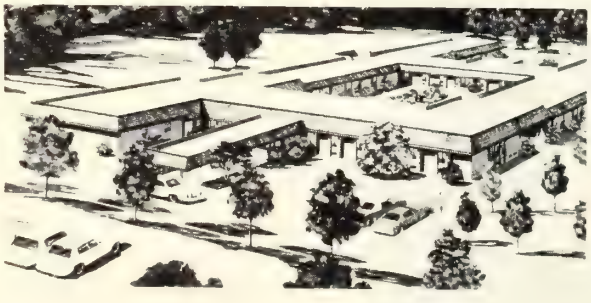

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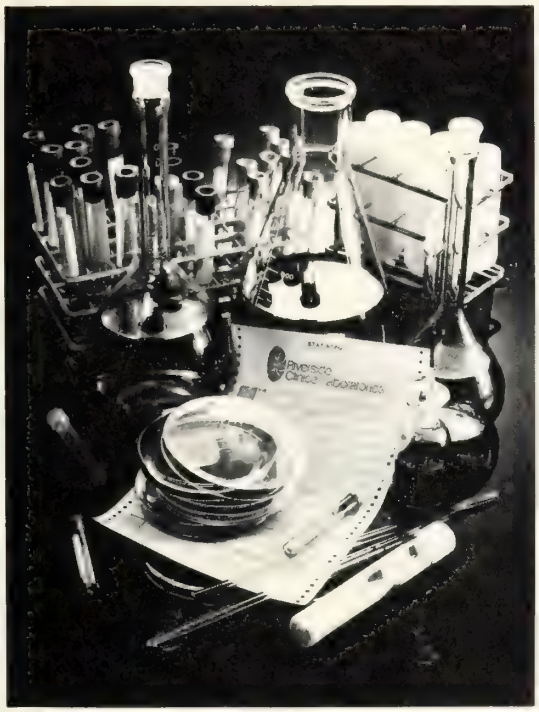

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VIRGINIA
MEDICAL
MARCH 1983

Stroke Mortality and Salt Ingestion in Eastern Virginia

George Moore, MD, *Warsaw, Virginia*

In the Eastern region of Virginia, around Chesapeake Bay, stroke is a predominant cause of death, especially intracerebral hemorrhage. Analysis of the data points to residency in rural areas of the region as a primary risk factor in this mortality and implicates dietary salt and high-sodium well water as contributors to essential hypertension and stroke.

Not without reason is the news media today labeling salt as the "silent killer." Dietary sodium is now dominating the interest of persons concerned with hypertension. Evidence of an association between sodium intake and hypertension has accumulated from epidemiological, animal and clinical studies. From animal studies, evidence is direct and unequivocal. The evidence in man has been indirect but supported by epidemiological research.

Whether sodium intake alone is responsible is not yet clear. Calcium and potassium as well as fats appear also to be significant factors as they interact with sodium. Those who challenge the argument that the general public should reduce salt intake in order to reduce the risk of hypertension point out the need for controlled human studies and realistic methods for measuring sodium intake. The press, however, is carrying the case to the public, and public response will no doubt force the issue.

This paper attempts to establish an association between salt ingestion and intracerebral hemorrhage mortality. A mortality indicator to estimate a risk outcome of salt ingestion would be useful and may help determine whether or not the issue of reducing sodium intake should be addressed aggressively by public sponsorship.

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Some of the information in this article was distributed to physicians in Eastern Virginia.

Submitted 5-25-82.

Stroke and Hypertension in Virginia

An indirect correlation of hypertension and stroke death rates can be inferred. The National Center for Health Statistics[1] has identified the Southeast as the nation's highest area of risk for cardiovascular disease and especially for stroke. Mortality rates are also higher in non-metropolitan areas and among blacks. Using age-adjusted mortality rates for cerebrovascular disease, the nation's death rate for black males aged 35-74 years in 1968-

Fig. 1. Eastern Virginia Planning Districts 17, 18, 21 and 22.

72 was 243.8 per 100,000. For Virginia, the mortality rate was 282.1.

The highest death rate in Virginia was found in the Southeast, or Tidewater, area, 333.9 per 100,000, and the lowest was in the area designated as Northern Virginia Metropolitan: 176.0. In the Tidewater area, a significant difference existed between Norfolk-Portsmouth, 336.2, and Newport News-Hampton, 255.5. Since the entire Tidewater area enjoys a high level of quality health care available to all regardless of means and place of residence, the argument that differences in health care can explain differences in stroke mortality among rural and urban populations may be controverted.

Hypertension data from the National Health Survey of 1971-75[2] showed higher mean systolic blood pressures of persons aged 25-74 years in the South, especially among blacks. Mean systolic blood pressures also were slightly higher among those who lived in rural areas and much higher among those with lower incomes and education. No unbiased hypertension data specific for Virginia were available at either federal or state levels.

Association of Salt, Hypertension and Stroke

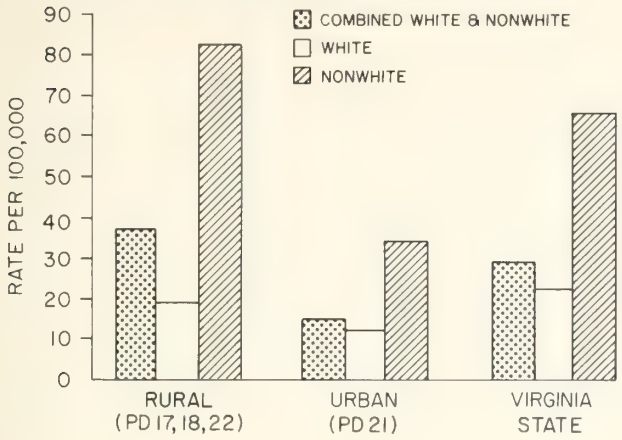
Hypertension has been long recognized as an important risk factor in the development of various diseases, including coronary heart disease, peripheral vascular disease, chronic renal failure and stroke, and the risk of death increases in direct proportion to blood pressure elevation.[3,4] Clinical trials[5] in the treatment of hypertension have demonstrated the efficacy of diuretics in reducing the incidence of stroke, especially intracerebral hemorrhage. Findings of the Framingham Study[6] have shown a linear relationship between levels of blood pressure and risk of stroke. In Japan, epidemiologic studies indicated that salt consumption, hypertension and stroke correlate significantly.[7,8] In Northern Japan, for example, where salt ingestion averaged over 26 grams a day up to the 1970s and hypertension prevalence exceeded 40%, stroke has been a leading cause of death. In the United States, where average salt intake is estimated at 10 to 15 grams per day, some 15%-20% of the population is believed to be hypertensive, with blacks exhibiting higher rates than whites for both hypertension and stroke mortality. In Africa, however, tribes not exposed to salt do not experience hypertension.[9,10] These studies suggest that differences in hypertension and stroke mortality among races may be due more to environmental than genetic factors.

Although hypertension has been recognized elsewhere[6,11,12] as the major risk factor for both occlusive cerebrovascular disease and intracerebral hemorrhage, most studies in the United States have been of limited value due to difficulties in localization of the vascular disease to cerebral infarction and hemorrhage. Nationally, 87% of all stroke deaths are due to cerebral infarction.[13] The Framingham study[14] gives the frequency of intracerebral hemorrhage as 5% of all strokes, and the Minnesota studies,[15,16] about 10%. The proportion of deaths from various causes of stroke varies considerably within other countries and does not usually correlate with atherosclerotic disease. Three patterns of stroke mortality in relation to coronary heart disease have been identified:[17] high death rates for both coronary artery disease and stroke, such as in Finland; high death rates for stroke and low death rates for coronary heart disease, such as in Japan; and low death rates for both diseases, as in Sweden. In each realized pattern, there was no sex difference within 45- to 74-year-old groups.

Japanese studies have been particularly valuable in helping to appraise the effects of salt-induced hypertension on stroke mortality. A 1968 study[18] of differences in the distribution of stroke lesions involving four-vessel angiography noted that the predominant lesions among Japanese were in the vertebral-basilar system and the cerebral arteries, while in a Minnesota population they were primarily at the carotid bifurcation. In addition, the Japanese exhibited high intracerebral arteriosclerotic disease of the circle of Willis, with aortic arteriosclerosis but low coronary atherosclerosis.

In a recent Japanese study,[19] it was found that the risk of intracerebral hemorrhage was associated

Fig. 2. Rates of Intracerebral Hemorrhage Mortality in Persons 55-64 by Place of Residence 1974-1978.



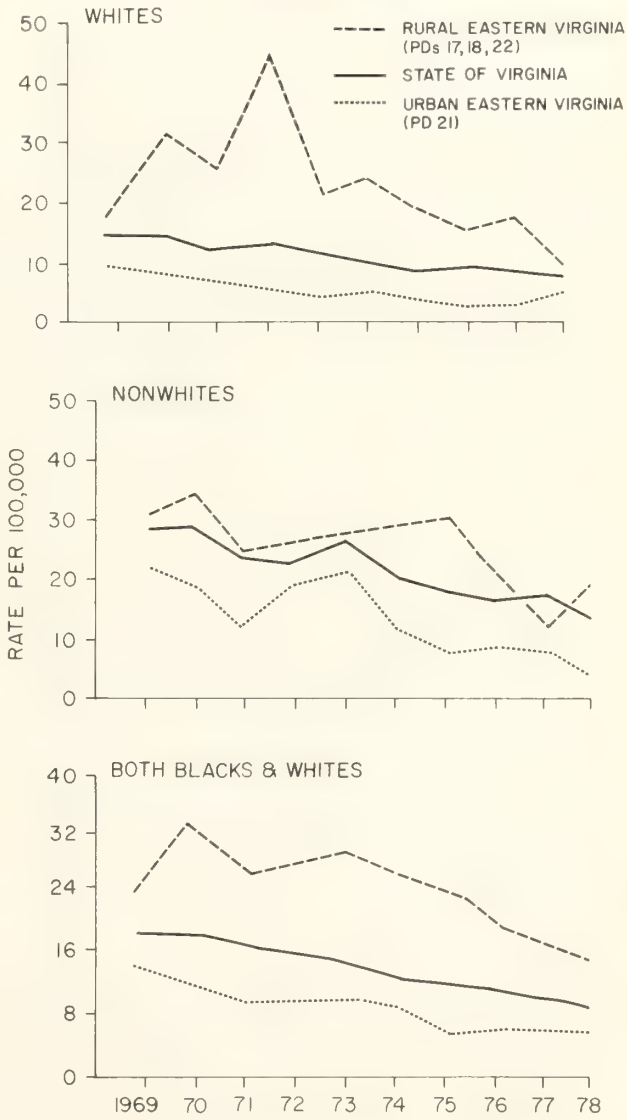
with low total serum cholesterol levels. As a risk factor for cerebral infarction, however, serum cholesterol held no weight. Since the same population experienced low mortality rates from coronary heart disease, it was concluded that a high salt, low animal protein diet constituted an essential risk factor for intracerebral hemorrhage.

The Westernization of Japanese diets is now resulting in higher serum cholesterol levels with a significant decline in the proportion of deaths from intracerebral hemorrhage among all age groups. This change has been most apparent among the higher socioeconomic classes. At the same time, mortality from all causes of stroke has declined tremendously.[20] The age-specific death rate for males aged 55-64 years has fallen 306 deaths per 100,000 from 1958 to 1976! The average salt intake of the Japanese is now estimated at 13 grams per day and probably less than 20 grams per day for Northern Japan. In the United States stroke mortality rates have been in decline since 1900 but have shown more than a 55% decrease since 1950.[21]

A few studies among blacks both in the United States and Africa are of related interest. A postmortem review[22] of cerebral atherosclerosis following deaths in Baltimore and Minneapolis showed that blacks in the Southern states have a greater degree of arteriosclerosis of the circle of Willis. Conversely, a study of stroke deaths in central Harlem[23] noted that the distribution by type among blacks was similar to that of the white population of New York City, while the case-fatality ratio within the area was also similar to that reported from predominantly white populations. It is now apparent that wide variations in racial mortality noted earlier are disappearing, and as rural blacks migrate to urban areas, high salt and low protein diets also tend to disappear. Remarkably, postmortem studies among Central Africans[22] have shown very little atherosclerosis of the circle of Willis as compared to both whites and blacks in the United States.

That extracranial atherosclerotic disease, so common among all United States whites and blacks of the South, is related more to the same risk factors as coronary heart disease is now quite evident. Intracranial disease as measured by deaths from intracerebral hemorrhage alone appears to point to the underlying problem of salt-induced hypertension uncomplicated by normal or high total serum cholesterol levels. The remote and rural Chesapeake Bay region would seem to have many of the characteristics required for a pertinent regional study of stroke mortality. In both rural Eastern

Fig. 3. Rates of Intracerebral Hemorrhage Mortality by Place of Residence 1969-1978.



Virginia and Northern Japan,[7,24] the classic diet up to the 1970s was primarily vegetarian, low animal fat, average protein, high carbohydrate and high salt.

Materials and Methods

The regional study area includes four of the State of Virginia's planning districts (Fig. 1): Northern Neck (Planning District 17), Middle Peninsula (PD 18), Peninsula (PD 21), and Eastern Shore (PD 22). Based on census information, these planning districts may be classified as either rural (PDs 17, 18 and 22) or urban (PD 21).

Mortality rates from 1969 through 1978 have been computed for all causes of stroke (ICDA 430-438) and intracerebral hemorrhage alone (ICDA 431). These rates have been analyzed for differences by age, race, sex, and place of residence. Rates for Virginia have also been computed as a basis for comparison. Age-specific intracerebral hemorrhage mortality rates among persons aged 55-64 years were used to analyze intraregional differences and to indicate salt-induced hypertension (Fig. 2). Trend lines were based on actual population estimates for each year, otherwise mortality rates were averaged for a five-year period. Age-adjusted death rates based on the census of 1940 were also scanned but not found as sensitive as age-specific rates in measuring the absolute levels of mortality necessary to track a disease process that appears to be in rapid decline. However, where associations were tested for statistical significance, both age-specific and age-adjusted rates were found comparable.

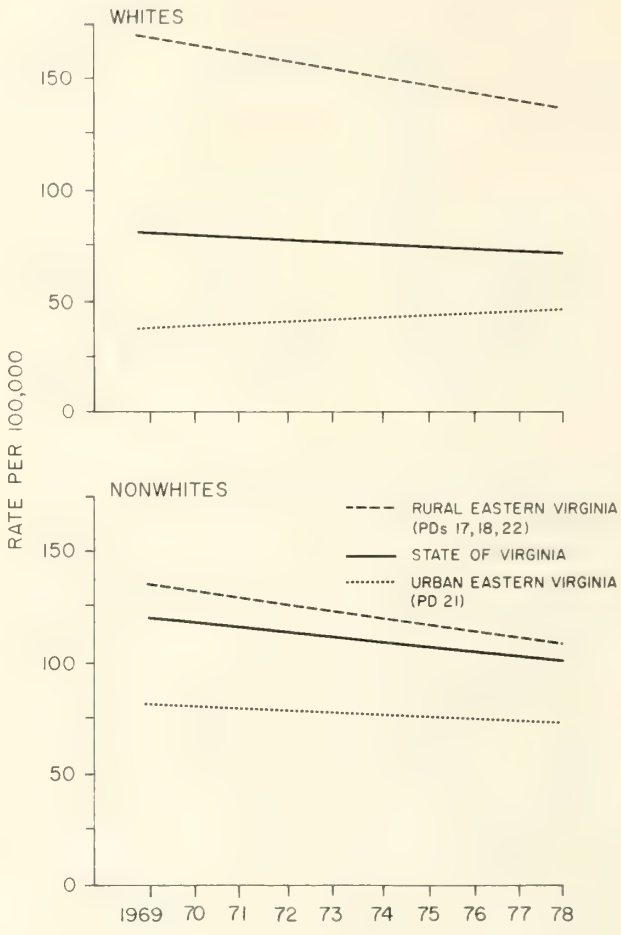
Results

While 13% of the nation's deaths from stroke are due to intracerebral hemorrhage,[13] the rate for the State of Virginia is 24%. Of all stroke mortality due to intracerebral hemorrhage in Eastern Virginia, 14% occurs in the urban area (PD 21) and 31% in the rural areas (PDs 17, 18, 22 combined). The rate among blacks in rural areas increases to 45%, approximating that of Northern Japan.

Age-specific rates for the aggregate of Eastern Virginia's rural planning districts are twice as high as the corresponding urban rate among both whites and blacks, with rural blacks again exhibiting the highest rate, four times higher than the rural rate for whites.

Trend lines for intracerebral hemorrhage mortality from 1969 through 1978 show that mortality over

Fig. 4. Rates of Cerebrovascular Disease Mortality by Place of Residence 1969-1978.



In Figures 2, 3, and 4, rates were computed from data from the Bureau of Vital Records and Health Statistics, Virginia Department of Health. Population data from the Virginia Department of Planning and Budget.

the State of Virginia has declined by 50% in this ten-year period, with a similar decline noted for the urban area of Eastern Virginia; mortality in rural Eastern Virginia, however, rose from 1969 to 1972 but has declined by about 60% from its 1972 peak (Fig. 3).

Analysis by race for deaths due to intracerebral hemorrhage indicates that trends for whites are quite different for rural and urban areas of Eastern Virginia (Fig. 3). The rural white rates have dropped significantly since 1972, although they have consistently been higher than state rates and those for urban PD 21. Among blacks, too, rates for the rural areas have consistently exceeded urban rates, but trends for both rural and urban blacks have been more alike than those among whites.

Unlike intracerebral hemorrhage alone, mortality

in Eastern Virginia from all causes of stroke among rural and urban blacks and rural whites has shown a steady decrease since 1969 (Fig. 4). Urban whites, however, exhibit an increasing rate for the ten-year period. The migration for retirement of older whites to the Peninsula area since 1960 may explain this trend. Analysis of 1980 United States Census data, when available, should offer evidence.

These data may also explain why stroke mortality rates are higher in Norfolk-Portsmouth than in adjacent Newport News-Hampton. We suspect that Norfolk-Portsmouth has attracted large numbers of low-income blacks from the rural counties of Eastern and Southside Virginia since World War I; these cities are heavily industrialized and offer jobs for unskilled labor. The growth of the Newport News-Hampton area, however, has been associated more with suburban development for the large number of military retirees and skilled workers who are drawn to the NASA and military industrial complex. In 1970, both of these urban areas held a black population of 28-30%, so that differences in stroke death rates noted today may be related more to place of birth than to place of death. If salt sensitivity originates in infancy and childhood as shown by animal studies[25,26] and is irreversible, then the place of death becomes somewhat incidental to the cause of death because of the mobility of the population under study. One can assume that the rural poor of the South were exposed to more dietary salt than others because of the availability of inexpensive pork and fish, and these were always preserved with salt for winter use. Refrigerators were slow in coming to the South and even those purchased in the 1940s and 1950s were used mainly for beverages.

As for the amount of sodium ingested from water, the entire population of the three rural Planning Districts 17, 18 and 22 consume ground water alone, so an analysis can be made by race and high- or low-sodium drinking water (Table 1). The black population of these districts have used shallow wells containing little or no sodium for generations. Since

Table 1. Intracerebral Hemorrhage Mortality Rates and Sodium Ingestion per 100,000 Persons Aged 20 and Over in Three Eastern Virginia Districts 1974–1978.

Area	Whites	Sodium Level in Well Water*	Non-Whites	Sodium Level in Well Water*
PD 17	19.0	High	16.0	Low
PD 18	19.7	High	26.1	Low
PD 22	12.5	Low	30.4	Low

* High = average sodium in deep well water 200–300 mgm/liter; low = average sodium in shallow well water 10–32 mgm/liter.

Table 2. Source and Intake of Sodium in Eastern Virginia.

Sources	Estimated Intake NaCl (gm/day)
Food: Naturally occurring in foods	2.5– 4.5
Added by industrial process	6.9– 8.4
Added by consumer (cooking, curing, table use)	6.0–10.0
Ground water	0.5– 3.0
Other sources: drugs, water softeners, etc.	1.0
Total	15.0–25.0

1930, whites in PDs 17 and 18 have increasingly used deep ground water, which contains high levels of sodium, but in PD 22, whites consume water from the same shallow-water aquifer as blacks. Thus in PD 22, the notably higher black rates can be presumed to be due to differences in dietary salt consumed. An estimate of sodium ingestion and sources of sodium intake in Eastern Virginia (Table 2) confirms that dietary salt ingestion should be highest among blacks of the rural PD 22.

In PD 17, white rates are higher than black rates and, in fact, are higher than white rates in PD 22. Rates among whites in PD 18, who also consume high-sodium well water, are also higher than in PD 22. These differences suggest that consumption of high-sodium ground water may be a significant factor in the higher risk of intracerebral hemorrhage experienced by whites in PDs 17 and 18. Tests of statistical significance for both age-adjusted and age-specific rates yield a p value of 0.025.

Discussion

This study of intracerebral hemorrhage mortality points to dietary salt burden as a major risk factor for stroke mortality in Eastern Virginia. Intracerebral hemorrhage mortality rates are higher in rural areas than urban areas. Rates for whites are lower than those for blacks in both rural and urban areas, but rural white rates are higher than urban white rates. The risk of stroke is highest for blacks in rural areas and lowest for whites in urban areas, where such rates for mortality approach the Framingham Study rates. The data resemble the pattern of stroke mortality in Northern Japan before 1970, when dietary salt intake was estimated at 26 to 30 grams daily.

Thus it can be hypothesized that dietary salt burden is a major risk factor for whites as it is for blacks, but most whites tended to remove themselves from high-salt foods several years before blacks.

Over several centuries, whites and blacks living together in Eastern Virginia consumed excess salt in food. In rural areas up to the 1940s, country stores supplied coffee, tea, sugar, flour and salt, but nearly all people, especially blacks, farmed, fished, crabbed and oystered. Hogs and fish were salted and cured for daily winter consumption. A visit today to country stores in PD 17, 18 and 22 shows that old customs still persist. Salt fish, fat back, pork hocks and sow belly are available, particularly in stores catering to blacks. Salt herring meals are advertised by churches. Fried pork skin, chitterling and tripe are sold as snacks in beer places. Commercial fishermen often have excess fish and will either give or sell these to employees for a few cents a pound, and quantities of fish are still preserved in barrels of brine for winter use. The taste of salt is easily acquired and carried over from generation to generation.

Urban migration, which began during World War I; electrification of rural areas in the 1940s with the introduction of the refrigerator; automobiles and shopping centers—all these changed food habits. Whites benefited from these changes first. Blacks followed, as installment purchasing and food stamps were introduced; according to an appliance dealer in PD 17, it was not until the 1960s that most blacks had purchased some type of refrigerator for their homes.

Black populations in rural areas have tended to remain stable in spite of loss of youth to the outside world, but white populations have changed remarkably since the peninsulas were opened by bridges, attracting a steady flow of retirees from metropolitan areas to waterfront properties. New homes have been built and new wells drilled. Deep artesian wells offered the "best" water for household use, but few recognized that this water contains up to 600 mgm of sodium per liter. Thus high-sodium drinking water has helped to rekindle old sodium sensitivities, particularly among the elderly who were exposed to excess dietary salt in infancy and childhood[25,26].

This has resulted in a curious situation. Trend lines of stroke mortality rates in these rural areas show that death rates are declining faster among blacks than whites and suggest that there will be little difference in stroke mortality rates among blacks and whites by 1990. It can also be assumed that as ingestion of animal fats increases in rural areas, the proportion of cerebral hemorrhage mortality to cerebral infarction mortality will decline.

Conclusion

In spite of the lack of definition of scientific evidence, it can be argued rather convincingly that stroke mortality is associated with excess dietary salt ingestion and that as salt consumption declines, stroke mortality falls. The picture we draw from our regional observation should apply elsewhere, since the dietary habits of Americans throughout the nation were similar before the refrigerator was introduced. National health policy[27] has been recently directed to the primary prevention of hypertension by means of advocating the reduction of salt intake among Americans by health promotion, labeling, and regulation of foodstuffs containing sodium. The national objectives for the 1980s include a reduction of average salt intake to 5 grams or less per day.

We would support the premise of a salt reduction policy now in the same way that scurvy was prevented on sailing vessels during the 16th century. Limes proved effective well before Vitamin C was identified in 1928. The difference in health care among groups of people might be helpful in explaining excess mortality from stroke in terms of secondary prevention, that is, after hypertensive disease can be detected clinically, but it does not relate to the primary prevention of hypertension.

Freis[28] and others have ventured to state that a general reduction of dietary salt below 2 grams per day would result in the prevention of essential hypertension in the United States and its disappearance as a major public health problem.

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Ru-Tuss Tablets are an oral antihistaminic, nasal decongestant and anti-secretory preparation.

INDICATIONS AND USAGE Ru-Tuss Tablets provide relief of the symptoms resulting from irritation of sinus, nasal and upper respiratory tract tissues. Phenylephrine and phenylpropanolamine combine to exert a vasoconstrictive and decongestive action while chlorpheniramine maleate decreases the symptoms of watering eyes, post nasal drip and sneezing which may be associated with an allergic-like response. The belladonna alkaloids, hyoscyamine, atropine and scopolamine further augment the anti-secretory activity of Ru-Tuss Tablets.

CONTRAINDICATIONS Hypersensitivity to antihistamines or sympathomimetics. Ru-Tuss Tablets are contraindicated in children under 12 years of age and in patients with glaucoma, bronchial asthma and women who are pregnant. Concomitant use of MAO inhibitors is contraindicated.

WARNINGS Ru-Tuss Tablets may cause drowsiness. Patients should be warned of the possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers.

PRECAUTIONS Ru-Tuss Tablets contain belladonna alkaloids, and must be administered with care to those patients with glaucoma, or urinary bladder neck obstruction. Caution should be exercised when Ru-Tuss Tablets are given to patients with hypertension, cardiac or peripheral vascular disease or hyperthyroidism. Patients should avoid driving a motor vehicle or operating dangerous machinery (See Warnings).

OVERDOSAGE Since the action of sustained release products may continue for as long as 12 hours, treatment of overdoses directed at reversing the effects of the drug and supporting the patient should be maintained for at least that length of time. Saline cathartics are useful for hastening evacuation of unreleased medication. In children and infants, antihistamine overdosage may produce convulsions and death.

ADVERSE REACTIONS Hypersensitivity reactions such as rash, urticaria, leukopenia, agranulocytosis, and thrombocytopenia may occur. Other adverse reactions to Ru-Tuss Tablets may be drowsiness, lassitude, giddiness, dryness of the mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerotomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, dizziness and insomnia. Large overdoses may cause tachypnea, delirium, fever, stupor, coma and respiratory failure.

DOSAGE AND ADMINISTRATION Adults and children over 12 years of age, one tablet morning and evening. Not recommended for children under 12 years of age. Tablets are to be swallowed whole.

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Pheniramine Maleate	20 mg
Pyrilamine Maleate	20 mg
Ammonium Chloride	200 mg
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Ru-Tuss Expectorant is an oral antitussive, antihistaminic, nasal decongestant and expectorant preparation.

INDICATIONS AND USAGE Ru-Tuss Expectorant is indicated for symptomatic relief of upper respiratory congestion associated with pharyngitis, tracheitis, bronchitis, and allergic rhinitis. Also, for the temporary relief of symptoms associated with hay fever, allergies, nasal congestion and cough due to the common cold.

CONTRAINDICATIONS Hypersensitivity to antihistamines. Concomitant use of an antihypertensive or antidepressant drug containing a monoamine oxidase inhibitor is contraindicated.

Ru-Tuss Expectorant is contraindicated in patients with glaucoma, bronchial asthma and in women who are pregnant.

WARNINGS Ru-Tuss Expectorant contains codeine phosphate, therefore, the patient should be warned of the potential that this drug may be habit forming. Ru-Tuss Expectorant may cause drowsiness. Patients should be warned of the possible additive effect caused by taking antihistamines with alcohol, hypnotics, sedatives and tranquilizers.

PRECAUTIONS Patients taking Ru-Tuss Expectorant should avoid driving a motor vehicle or operating dangerous machinery (See Warnings). Caution should be taken with patients having hypertension, diabetes, hyperthyroidism and cardiovascular disease.

Caution should also be used in patients with pulmonary, hepatic or renal insufficiency.

ADVERSE REACTIONS Ru-Tuss Expectorant may cause drowsiness, lassitude, giddiness, dryness of mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, and insomnia. Overdoses may cause restlessness, excitation, delirium, tremors, euphoria, metabolic acidosis, stupor, tachycardia and even convulsions.

DOSAGE AND ADMINISTRATION Adults: 1 or 2 teaspoonfuls, orally, every 4 hours, not to exceed 10 teaspoonfuls in any 24-hour period.

Children 6 to 12 years of age: ½ the adult dose, not to exceed 6 teaspoonfuls in any 24-hour period. Children 2 to 6 years of age: ½ teaspoonful every 4 hours, not no exceed 3 teaspoonfuls in any 24-hour period. Children under 2 years of age: Use as directed by a physician.

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Fallopian Tube Microsurgery

Bruce G. Bateman, MD, Wallace C. Nunley, Jr., MD, and
James D. Kitchin III, MD, *Charlottesville, Virginia*

At the University of Virginia, microsurgery has significantly improved some tubal plastic procedures. Particular success has been achieved in restoring fertility to the increasing number of patients requesting reversal of previous ligations.

In recent years the University of Virginia Medical Center has had a growing number of referrals for reconstructive surgery of the oviduct. Patients requesting reversal of sterilization have frequently had tubal ligations performed at a relatively young age. Many have had a change in marital status. Others have lost children due to accident or illness. Some have simply changed their minds. Using microsurgical techniques, we have generally been highly successful in restoring the fertility of these patients.

We have also seen a great many patients with inflammatory obstruction of the fallopian tube and have used microsurgery in treating them. Inflammatory obstruction may be caused by gonococcal pelvic inflammatory disease, non-gonococcal pelvic inflammatory disease (possibly IUD-related), appendicitis/appendectomy or any pelvic surgical procedure (oophorocystectomy, salpingectomy). A strikingly high percentage of our patients have had previous abdominal or pelvic surgery. Our success in restoring tubal patency and fertility in this group of patients has been highly variable and has depended largely on the severity of their disease.

From the Department of Obstetrics and Gynecology, University of Virginia School of Medicine. Address correspondence to Dr. Bateman at Box 387, University of Virginia School of Medicine, Charlottesville VA 22908.

Submitted 2-17-82.

Microsurgery in Gynecology

The application of microsurgical techniques to gynecology has improved the results of tubal plastic surgery, with the most significant improvement in the reversal of tubal sterilization. Procedures to correct pathologic obstruction of the fallopian tubes have shown similar but less dramatic improvement.

The use of magnification to perform tubal anastomosis offers the advantage of allowing for precise suture placement in relatively small structures (diameter of the proximal tubal isthmus = .05 mm). It also allows for pinpoint hemostasis to minimize tissue damage. The principle postoperative complication of tubal anastomosis is ectopic pregnancy.

In 1956 Siegler and Hellman reported the results of 191 tubal anastomosis procedures performed by 53 surgeons using macrosurgical techniques;[1] the success rate was 7.8%. In comparison, microsurgical techniques in tubal anastomosis have resulted in

pregnancy rates ranging from 58% to 64%.[2–4] In a 47-patient subgroup of Gomel's series, with at least an 18-month followup, a pregnancy rate of 80% was documented.[4] Ectopic pregnancy rates vary from 1.3–13%.[2,3]

Sibler and Cohen, reporting on the effect of tubal length on the fertility of patients undergoing tubal anastomosis, found no pregnancies in patients with postoperative tubal length less than 3 cm, 45% pregnancy rate in patients with 3–4 cm, and 100% pregnancy rate in patients with greater than 4 cm.[5] Another success factor may be the length of time between tubal ligation and anastomosis. Winston observed a 50% reduction in the success of anastomosis procedures performed five years or more after the original surgery.[4] Vasquez and coworkers found electron microscopy evidence of flattening of tubal epithelium, loss of cilated cells and atrophy of tubal mucosa in patients who were more than four years post surgery.[6] The significance and reversibility of these changes is unknown.

Tubal damage due to inflammatory obstruction varies in severity. The terminology applied to the surgical correction of this condition is poorly standardized, and comparison of reported data is difficult. Siegler and Kontopoulous compared the results of conventional surgery and microsurgery related to fimbrioplasty (fimbria present with partial or complete obstruction) and salpingostomy (fimbria absent with tubal occlusion).[7] They found an improvement in the term pregnancy rate with microsurgery.

Winston reported an improvement in the results of salpingostomy from a 9% pregnancy rate with conventional surgery to 17.5% pregnancy rate with microsurgery.[4] Winston and others have noted an increase in ectopic pregnancy rates with the use of microsurgery; the cause of this is unknown but may be related to improved postoperative patency in badly damaged tubes. The Mulligan hood (a Silastic® prosthesis) is preferred by a few surgeons to correct terminal fallopian tube obstruction; a major drawback related to its use is the necessity for a second operative procedure for removal.

Another facet in the postoperative management of patients with distal tubal obstruction is hydrotubation, or flushing a liquid medium through the fallopian tubes. In 1971 Grant reported an increase in term pregnancy rate with the use of postoperative hydrotubation using hydrocortisone or chymotrypsin.[8] Since that report the procedure has gained popularity, and a variety of media have been used, including antibiotics, saline, dextran, proteolytic enzymes and corticosteroids.

Table 1. Comparison or Results of Macrosurgical and Microsurgical Techniques.

	Macrosurgery			Microsurgery		
	Number of Patients	% Term Pregnancy	% Ectopic Pregnancy	Number of Patients	% Term Pregnancy	% Ectopic Pregnancy
Salpingostomy	15	13	20	23	26	13
Fimbrioplasty	11	18	18	18	56	11

Obstruction at the uterine cornu may be due to infection, salpingitis isthmica nodosa or tubal sterilization. There are several techniques for reimplantation of the distal fallopian tube in cases of cornual obstruction, including cornual resection with the scalpel or reamer followed by reimplantation; cornual resection with transfundal incision and reimplantation; posterior fundal incision and reimplantation. These procedures have produced pregnancy rates varying from 9–50% with no one procedure showing consistently superior results.[9,10] Transfundal incision is performed to allow direct visualization of the tubal implantation, and some surgeons prefer to leave a stent in place during the immediate postoperative period. Stents of this type can be removed through the cervix. With the resection of the uterine cornu or transfundal uterine incision there is an increased risk of uterine rupture with subsequent labor, and delivery by Cesarean section is performed in these patients.

In 1977 Winston reported a 69% pregnancy rate in a group of 16 patients with cornual obstruction by performing a microsurgical anastomosis of the distal tube to the interstitial tube.[11] Gomel has also reported improved results using this technique.[3] Since the uterine wall remains intact, patients undergoing this procedure do not require delivery by Cesarean section.

Various medical adjuvants are in use in conjunction with microsurgery, including systemic glucocorticoids, promethazine, antibiotics, and intraperitoneal dextran. These adjuvants are intended to reduce or modify inflammatory response and therefore diminish adhesion formation. Their use is based primarily on animal experimentation and their value has not been comfirmed in controlled human studies.

Tubal Surgery at the University of Virginia

At the University of Virginia Medical Center all gynecologic microsurgery procedures are performed by a member of the Endocrine Infertility

Division. Preoperative evaluation of patients requesting reversal of sterilization includes a history and physical exam and review of the previous operative note when available. We perform a preoperative hysterosalpingogram in order to assess the uterine cavity and proximal fallopian tube. The operating surgeon has a thorough discussion with the patient and her husband to ensure their understanding of the procedure, including failure rate, risks of tubal pregnancy, and necessity for Cesarean section if reimplantation is performed.

Patients have a one-stage procedure. Laparoscopy is performed to assess the amount of remaining fallopian tube. If conditions are favorable for anastomosis, a laparotomy is then undertaken. For magnification we use the Zeiss operating microscope. Lysis of adhesions and mobilization of the fallopian tube segments is carried out with needlepoint electrocautery. The occluded ends of the fallopian tubes are sharply resected and tubal anastomosis is performed using 7–0 coated polyglycolic acid (Vicryl®).

The results of our tubal anastomosis procedures have been encouraging. Postoperative tubal patency in this group of patients is virtually 100%, and the pregnancy rate is similar to that of Gomel and Winston.[3,4] A few patients have conceived on the first cycle following resumption of intercourse. There have been very few ectopic pregnancies in our series.

If cornual anastomosis is not possible due to complete obstruction of the interstitial fallopian tube, a reimplantation is performed. The interstitial tube is excised and the distal segment reimplanted. (We do not use stents and consider transfundal uterine incision unnecessary). Not all aspects of tubal reimplantation require magnification, and we have found Loupes glasses useful. Loupes glasses magnify 4.5 to 6 times.

Distal fallopian tube obstruction requires fimbrioplasty or salpingostomy. If a dimple is noted in the vicinity of the fimbria the tube is teased open using blunt dissection. Areas between the mucosal folds of the endosalpinx are then sharply incised or divided with needlepoint electrocautery. Loupes glasses or the operating microscope are used to identify these areas and avoid unnecessary injury to healthy mucosa or blood vessels. The tubal ostium is recreated with 7-0 Vicryl®. Care is taken to avoid suturing the tubal mucosa. If complete obstruction with hydrosalpinx formation is encountered, the distal tube is sharply incised and the incisions extended radially between mucosal folds to recreate the ostium as described above.

Cases requiring extensive surgery due to pelvic adhesions usually result in raw areas on the uterus, broad ligament, or pelvic pertineum. In this situation we use a free peritoneal graft, taken from the anterior peritoneum, to cover raw surface areas and prevent adhesion reformation. In addition a tubopexy may be performed, fixing the tube by a single fine suture to the pelvic sidewall. Patients having surgery for distal tubal obstruction are treated with hydrotubation on the third and fifth postoperative days and then return during the second and third postoperative weeks for two additional treatments; 6% dextran 70 is used and causes a minimum of discomfort.

Patients undergoing all types of tuboplasty are treated with intraoperative dexamethasone and penicillin followed by a three-day postoperative course unless there is a history of drug allergy or other contraindication. In our experience, dexamethasone therapy has not interfered with wound healing or promoted infectious morbidity. It has the pleasant side effect of affording patients a sense of well being, and most patients require a minimum of narcotic analgesics.

Our results in patients with distal tubal obstruction have correlated closely with the severity of disease. Patients with minimal tubal dilatation have had the best prognosis, with pregnancy rates in the range of 35-40%. Patients with badly dilated tubes have had a rather poor prognosis for postoperative tubal patency and fertility. We have observed an ectopic pregnancy rate similar to that reported by Diamond and Gomel.[2,3]

We perform a hysterosalpingogram two to three months after surgery to assess tubal patency. If tubal patency is documented but the patient fails to conceive within one year, a second-look laparoscopy may be recommended. Small but significant peritubal adhesions may be divided through the laparoscope without a second major surgery. In cases of complete reocclusion, major surgery is not recommended due to the very low success rate of repeat tubal plastic procedures.

A striking number of our patients have a history of previous pelvic surgery or appendectomy. Therefore, we have chosen to apply some of the principles of microsurgery to routine pelvic procedures performed on young women in whom preservation of fertility is a consideration. In an effort to minimize postoperative adhesion formation, we use fine, non-reactive suture, free peritoneal grafts where indicated and, in some cases, intraperitoneal dextran.

concluded on page 176

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1. National Interagency Council on Smoking and Health. *The Smoking Digest.* Progress Report on a Nation Kicking the Habit. 1977

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continued from page 171

Summary

The principles of microsurgery have significantly improved the results of some tubal plastic procedures. This implies not only the use of magnification but the observation of surgical techniques intended to minimize tissue trauma and diminish adhesion formation. Medical adjuvants may be of value in this regard as well.

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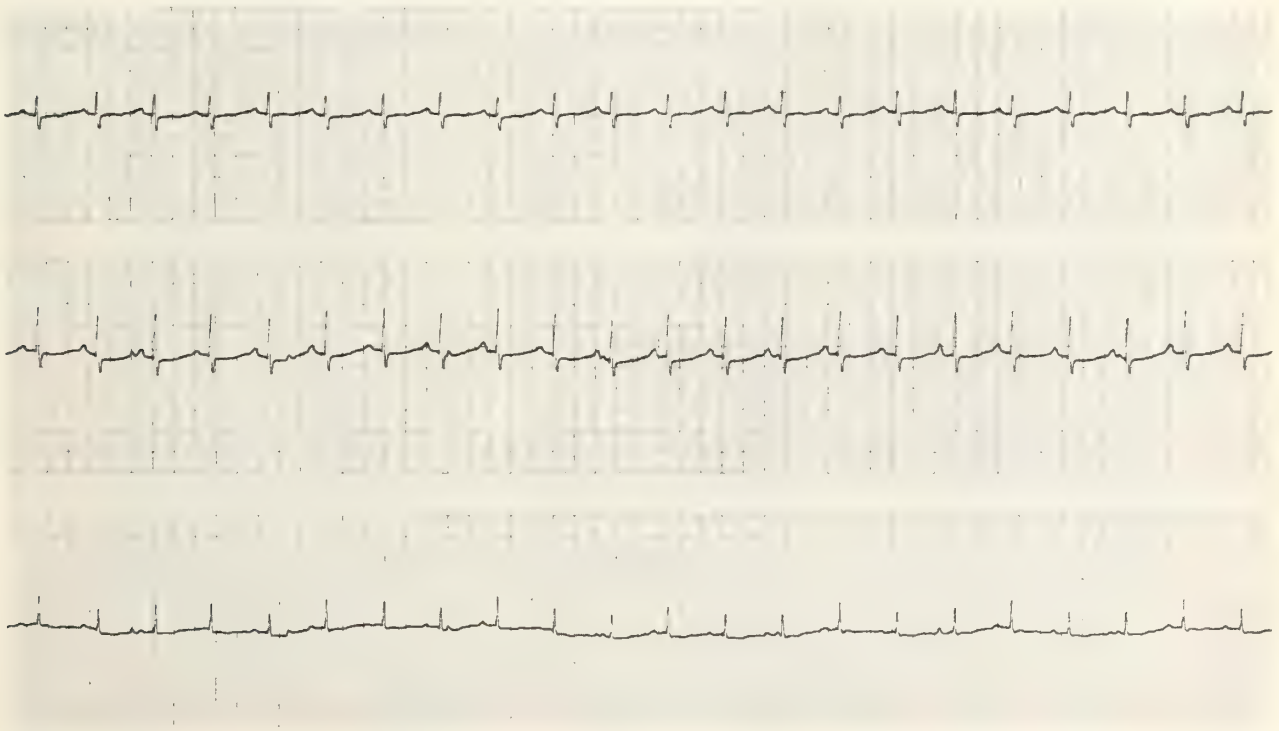
Can You Diagnose This?

A medical puzzle prepared by
Mikel D. Smith, MD, and Donald W. Romhilt, MD, *Richmond, Virginia*

A 63-year-old woman with a long history of rheumatoid arthritis with restrictive lung disease and bronchiectasis was hospitalized for symptoms of increasing dyspnea and orthopnea. This rhythm strip of leads I, II and III was obtained shortly after admission. What was the diagnosis? *From the Department of Medicine, Division of Cardiology, Medical College of Virginia.*



DIAGNOSIS/DISCUSSION NEXT PAGE

Diagnosis

Sinus tachycardia. Atrial dissociation.

Discussion

In leads II and III there are small positive deflections after the second QRS complex just prior to the third P wave; after the fifth QRS complex and before the sixth P wave; just after the eighth QRS complex, between the eleventh P wave and QRS complex, between the fourteenth P wave and QRS complex, on top of the seventeenth P wave changing its configuration, and between the twentieth P wave and QRS complex. The ectopic atrial rhythm has a rate of 38 to 40 and is slightly irregular.

Atrial dissociation is a rare electrocardiographic finding characterized by a unilateral ectopic atrial rhythm independent of the basic rhythm. Thus there are two independent P waves seen on the surface EKG. The ectopic focus appears as small, bizarre P′ waves at a rate of 30-60 per minute which do not interfere with the sinus mechanism and are not conducted to the ventricles. The P′ waves are often followed by a series of low-amplitude, high-frequency oscillations called "microfibrillatory waves" lasting less than one second. The P′-P′ interval may be slightly irregular. Rarely, the unilateral atrial impulses may be atrial tachycardia, atrial fibrillation or even atrial flutter.

Atrial dissociation is thought to occur when an isolated area of atrial tissue exhibits entrance and exit block. There appears to be interruption of impulse conduction in a part of the atrium, while the isolated section of atrial tissue has its own autonomous pacemaker. The fine oscillations following the P′ wave may represent fibrillation in the localized area of atrium. Despite numerous case reports and experimental reproduction of the arrhythmia, its existence has been questioned. Some authors have suggested the ectopic P waves represent the recording of action potentials from the diaphragm or accessory muscles of respiration.

The differential diagnosis of this EKG finding includes atrial parasystole, cardiac transplantation, atrial premature beats and various artifacts.

Atrial dissociation is almost always found in the clinical setting of severe, intractable congestive heart failure and often occurs just prior to death. It has also been reported in various other conditions including myocardial infarction, glomerulonephritis, digitalis intoxication, diphtheria and congenital heart disease.

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Antiglobulins as Diagnostic Aids in Endocarditis

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Numerous reports have associated antiglobulins with infections, particularly endocarditis, but in this study the correlation of positive tests for antiglobulins with endocarditis was poor. The authors' data suggest that such tests usually are not specific enough to be of use in diagnosing this disease.

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Tests for anti-Fc antiglobulin antibodies (rheumatoid factors) are often requested for patients suspected of having bacterial endocarditis based on the reported association of these antiglobulins with this disease.[1-3] These antiglobulins have been demonstrated in a variety of diseases and are found even in normal individuals, especially in the aged, although titers comparable to those seen in rheumatoid arthritis are rarely encountered.[4-6]

A second group of antiglobulins known as serum agglultinators or homoreactants, when present in high titer, are known to be associated with severe supportive infections.[7,8] These anti-Fab antiglobulins were first described by Osterland et al, who found them in most human sera and identified them as IgG antibodies directed against antigenic determinants on the Fab fragments from enzymatically digested molecules of IgG.[9] Subsequent studies have shown that a multiplicity of agglutinators exist in human sera and that their specificities are determined by modifications of Fab fragments characteristic of the particular enzymes used for hydrolysis, e.g., pepsin, papain, subtilisin, trypsin, etc.[10-12] All of the agglutinators tend to rise in titer in severe and protracted suppurative infections, and with resolution of the infections the titers fall, usually with a concomitant reduction in the levels of gamma globulin.[7]

These two antiglobulins are unrelated, and high titers are characteristically associated with different states of disease. The etiology and pathogenetic significance of elevated titers are poorly understood.

This study was undertaken to determine the value of these two antiglobulin tests as diagnostic aids in patients with bacterial endocarditis. In addition, tests for C1q binding by soluble immune complexes, serum levels of C3 and C4 and gamma globulin responses were performed on the patients with endocarditis to determine the correlation (or lack of it) with the antiglobulin responses.

Materials and Methods

In this study, the patient population was divided into two groups, depending upon where initiation for test performance originated. In Group 1, the blood samples were sent to the laboratory for a test for serum agglutinators in patients with endocarditis. Thus, the clinical condition of the patient determined the request for the test.

In Group 2, positive tests for serum agglutinators were found following routine screening of all bloods sent to the laboratory for tests for rheumatoid factors. These tests for rheumatoid factors were

usually requested by physicians associated with the Division of Immunology and Connective Tissue Disease but were also requested by others when the disease process was obscure or when an autoimmune process was suspected. In addition, a request for theumatoid factors is believed to be helpful as a diagnostic aid in patients suspected of having subacute bacterial endocarditis.

Rheumatoid Factors. All serum samples were tested for rheumatoid factors by three tests selected for increasing specificity: slide latex test, sensitized human cell test (SHC), and sensitized sheep cell test (SCC).[6] In this study, a test for rheumatoid factors was considered positive if two of these three tests were positive and if the titer was 1:160 or above by the sensitized human cell test. However, positive tests for rheumatoid factors as determined by only the latex slide test and/or SHC test with a titer of less than 1:160 were recorded for comparison with previously published results. The sensitized sheep cell test as performed in this laboratory is not as sensitive as that used by other laboratories. It is standardized so that it is very rarely positive in the absence of rheumatoid arthritis, the disease characterized by the highest titers of these antiglobulins. Rheumatoid arthritis is also used as the determinant for standardization of commercial reagents.[13]

Serum Agglutinators. All serum samples were tested for two serum agglutinators, usually papain and subtilisin agglutinators. If these gave a strongly positive test at a serum dilution of 1:20, the serum was tested for all four serum agglutinators, i.e., papain, subtilisin, chymotrypsin and trypsin agglutinators, and titrations performed. A test for serum agglutinators was considered positive if three of these four tests were positive with a titer of 160 or above. Methods for performing these tests have been reported in detail elsewhere.[10-12]

^{125}I-C1q Binding Assay for Detection of Circulating Immune Complexes. The procedure of Nydegger et al[14] was modified and consisted of incubating 50 μl of test serum with 100 μl of Na_2EDTA 0.12 M, pH 7.5, for 30 minutes at 37°C, with transfer to an ice bath. Fifty microliters of ^{125}I-C1q and 1 ml of 2.2% polyethelene glycol were added with continued incubation in an ice bath for 60 minutes, followed by centrifugation at 1500 g for 20 minutes at 4°C. The precipitate was counted and the C1q binding activity derived from percentage ^{125}I-C1q precipitated as compared with the radioactivity precipitated in a trichloracetic acid (TCA) control tube in which 1 ml 20% TCA was added to 100 μl of bovine albumin mixed with 50 μl of ^{125}I-C1q. Tests were run in duplicate. Controls consisted of heat-aggregated gamma globulin at varying concentrations and a normal serum. The lower limit of sensitivity of the test was 50 μg of aggregated human globulin per ml serum, and the coefficient of variation of the test 3.5%.

Table 1. Incidence of Positive Tests for Serum Agglutinators and Rheumatoid Factors in Two Populations.

Study Group	Total Patients	Serum Agglutinators		Rheumatoid Factors	
		Number Positive	% Positive	Number Positive	% Positive
Group 1. Endocarditis	34*	7	20.5	9	26.4
Group 2. Hospital Population	3898**	21	.005	1,130	29.0

* All patients had clinical diagnosis of endocarditis
** Blood samples submitted for tests for rheumatoid factors were tested for serum agglutinators

Complement Component Measurements. Levels of serum C3 and C4 were measured by radial immuno-diffusion as described by Ruddy and Austen.[15]

Results

Table I shows the results of tests for serum agglutinators (anti-Fab IgG) and rheumatoid factors (anti-Fc IgG) in the two study groups. Although the incidence of positive tests for rheumatoid factors are similar, the group with endocarditis showed over a 4000-fold increase in posistive tests for serum agglutinators. When only the patients with endocarditis are considered, we find that there is little difference in the incidence of the two antiglobulins.

Among the 21 individuals from the hospital population with positive tests for serum agglutinators, 15 had demonstrable infections, thus, correlation with infection was 71.4%. This contrasts with the endocarditis group where correlation with infection was only 20%. As noted, only 26% of the endocarditis patients had positive rheumatoid factor tests, and none had rheumatoid arthritis.

The 34 blood samples from patients with endocarditis were sent to the laboratory for tests for serum agglutinators. In addition, the test for rheumatoid factors, C_3 and C_4, C1q binding assay for immune complexes, and electrophoretic patterns were performed. Because only seven patients gave positive tests for serum agglutinators, samples were obtained later in the patients' hospitalization, and a few become positive. However, since this study was to evaluate these antiglobulin antibodies as diagnostic aids, only the initial sample was consid-

ered. Five of the nine positive tests for rheumatoid factors were only weakly positive slide tests. When these results were evaluated according to the criteria established at the onset of this study (positive slide test and SHC test = 160), there were no positive tests.

The C1q binding assay for soluble immune complexes showed that 12 of the patients (38%) had elevated levels. The levels of gamma globulin were not strikingly elevated, and although 14 patients (41%) showed values in excess of normal (20 ± 4), only one patient showed twice the normal value. Significant depression of C3 or C4 values was uncommon. Statistical evaluation showed no significant correlation between any of these tests and the serological responses as measured by the antiglobulins.

Discussion

Numerous studies have documented the association of rheumatoid factors with bacterial endocarditis. Williams and Kunkel found that 50% of the patients they studied showed positive latex tests.[3] Bacon and colleages found rheumatoid factors in 70% of their patients and anti-nuclear antibodies in 61%. These authors reported a high incidence of candida antibodies, anti-smooth muscle antibodies and elevated gamma globulin levels.[1] Others confirmed the high incidence of latex reactions, especially when associated with chronicity of infection and elevated levels of gamma globulin.[2,16] However, none of these investigators showed a control group of hospitalized patients tested with the same reagents.

In quest of the correlation between diagnosis and results of serologic tests for antiglobulin antibodies, a report from this laboratory indicated that among 181 patients hospitalized with a variety of diseases, SHC tests with a titer of 160 or above were positive in 7.1%. This value was much higher if latex slide tests were accepted as definitive positive tests (16.5%).[17] Commercially available latex reagents have been observed to vary in sensitivity over the years. A pertinent control population is therefore essential in evaluating the diagnostic usefulness of these tests. It is imperative that the control population be from the same group from which patients with a specific disease are selected. Thus, using a group of normal individuals as a control population for a study of the value of rheumatoid factors in endocarditis would lead to a false impression of the value of these as diagnostic tests. In order for a test to function as a diagnostic aid, there must be a good correlation between the positive test and the disease for which it is ordered and the absence of such correlation with other disease processes in the same hospital population. According to these standards neither of the antiglobulin tests evaluated in this study functioned well as a diagnostic aid for endocarditis.

Correlation of positive tests for serum agglutinators and infection was influenced by method of selection of test subjects. In Group 1, the patients with endocarditis were selected and then the test was performed. In Group 2, we performed the tests, selected those with positive tests and then determined the state of disease. In Group 1, the test results are influenced by the duration of the infection, the immunocompetence of the host and certain characteristics of the pathogen. In Group 2, these qualities are excluded as we are only considering those with positive tests.

Probably one of the reasons that tests for rheumatoid factors are such effective diagnostic aids in rheumatoid arthritis is that they are usually determined in patients who are members of an ambulatory population with absence of other significant systemic diseases. In addition, it is certainly true that in this disease we consistently find the highest titers of these antiglobulins, and thus, tests that are made relatively insensitive are still valuable as diagnostic aids.

Summary

We conclude from our studies that neither antiglobulin test will serve as a satisfactory diagnostic test in endocarditis, but when bloods are screened for serum agglutinators, positive tests may be taken as strong evidence that infections are present. Although both antiglobulins are stimulated by diseases characterized by inflammation, the fact that the correlation between the two antiglobulins is poor suggests that different immunologic facets influence their production.

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Pheochromocytoma as Cause of Hypertension after Coronary Bypass

Edward A. Lefrak, MD,
Quentin MacManus, MD, and
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TRANSIENT postoperative systemic hypertension occurs in nearly one-third of patients undergoing coronary artery bypass grafting. Studies in such patients incriminate varying mechanisms, including disturbed neurogenic reflexes, release of catecholamines, and hyperactivity of the renin-angiotensin system.[1-5] Regardless of etiology, the hypertension generally disappears in three to 36 hours. We recently encountered a patient in whom profound hypertension unexpectedly persisted following initially uncomplicated coronary artery bypass. The discovery of a large pheochromocytoma was both instructive and humbling.

Case Report

A 71-year-old man was evaluated for progressive, disabling angina pectoris. He had suffered an acute myocardial infarction several years previously. There was no history of hypertension. In addition to exercise-induced symptoms of angina, the patient reported paroxysms of dyspnea, headache and diaphoresis, occasionally accompanied by anginal chest pain when he bent over while gardening. On the advice of his physician, the patient measured his blood pressure during several such attacks but never found it to be elevated. His angina pectoris, which occurred more frequently than the paroxysms, progressed until it considerably limited his daily activities. He was treated with propanolol, 160 mg per day, with little change in clinical status. A stress test was positive. Coronary angiography demonstrated triple vessel atherosclerotic heart disease with good left ventricular function. He underwent double coronary artery bypass at the Fairfax Hospital on October 21, 1980. The operation proceeded uneventfully.

During the early postoperative period, systemic blood pressure was elevated, requiring treatment with nitroprusside. Unusually wide and sudden fluctuations of the systolic blood pressure from 50 to 200 mm Hg were observed. On the day following surgery, frequent premature ventricular contractions required both lidocaine and procainamide for control. Sinus tachycardia persisted.

Two days postoperatively the upper half of the patient's body appeared flushed. He became disoriented, agitated and diaphoretic and complained of headache. Propanolol was added without apparent effect on systolic pressure. A focal seizure occured during a blood pressure nadir.

Seven days after surgery, a 12-hour urine specimen contained 2817 mcg catecholamines (normal = 0-100 mcg), 83% norepinephrine and 17% epinephrine, and 16.6 mg metanephrines (normal = <1). Computerized axial tomography demonstrated a 6-cm, right adrenal mass (Fig. 1). The blood pressure was stabilized on 22 mg per day of phenoxybenzamine in preparation for reoperation. A pheochromocytoma 6 × 4 × 3.5 cm and weighing 70 grams was excised on 12/1/80 (Fig. 2). The postoperative course was uneventful. He has resumed full activity and has remained normotensive without medications.

Discussion

Ninety percent of patients with pheochromocytoma report paroxysmal symptoms, most commonly headache, palpitations and diaphoresis, and 90% have elevated blood pressure, although in one-third the hypertension is episodic. Our patient experienced typical paroxysms but did not demonstrate sustained hypertension. Blood pressure during the paroxysms was normal, and thus laboratory tests for pheochromocytoma were not performed. The patient's subsequent post-coronary artery bypass course of rapidly fluctuating blood pressure and increased levels of catecholamines suggest that he had measured his blood pressure too long after the

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onset of symptoms to demonstrate hypertension. As virtually all patients with pheochromocytoma have elevated urinary catecholamines and their metabolites, this case illustrates the usefulness of such tests in patients with classical paroxysms of pheochromocytoma even though sustained hypertension is absent.

Chest pain is part of the paroxysms in 20% of patients with a pheochromocytoma. These patients may be predisposed to accelerated coronary atherosclerosis, producing angina. Coronary artery vasospasm or increased alterload caused by elevated levels of circulating catecholamines from the pheochromocytoma may exacerbate the angina syndrome.

Although hypertension is common after coronary artery bypass grafting, sudden paroxysms of blood pressure to 200 mm Hg, or persistence of marked hypertension associated with gastrointestinal symptoms, or hyperglycemia beyond the first few postoperative days should be suspect. Normal catecholamine determinations are useful to rule out the possibility of a pheochromocytoma producing hypertension, but elevated levels may be difficult to interpret in the first few days after an operation.[6] Roberts et al measured plasma norepinephrine and epinephrine soon after coronary artery bypass[4] and found that catecholamines were higher in hypertensive than normotensive patients; however, the results were not specific because the levels were elevated in both groups subjected to the stress of surgery. The phentolamine (Regitine®) test for diagnosis of pheochromocytoma is rapid, but the profound hypotension that it occasionally elicits may be hazardous in the recovery period after cardiac surgery; also, the test is associated with frequent false-positive and -negative results, the latter illustrated by the failure of our patient's blood pressure to respond to this drug.

A rapid and reliable method for diagnosis of a pheochromocytoma is computerized tomography.[7,8] The technique detects adrenal tumors greater than one cm in diameter. The expense and radiation dose preclude its use as a screening test for pheochromocytoma; however, when the index of suspicion is reasonably high and clinical considerations mandate rapid diagnosis, CT scan is the diagnostic procedure of choice.

When a pheochromocytoma is diagnosed and localized by computerized tomography, surgical extirpation may be planned. The patient is prepared with intravascular volume repletion and catecholamine blockade. Blockade has generally been accomplished at the alpha adrenergic receptor level

Fig. 1. Abdominal CT scan showing supra renal pheochromocytoma (arrow). 1 = inferior vena cava. 2 = right kidney.

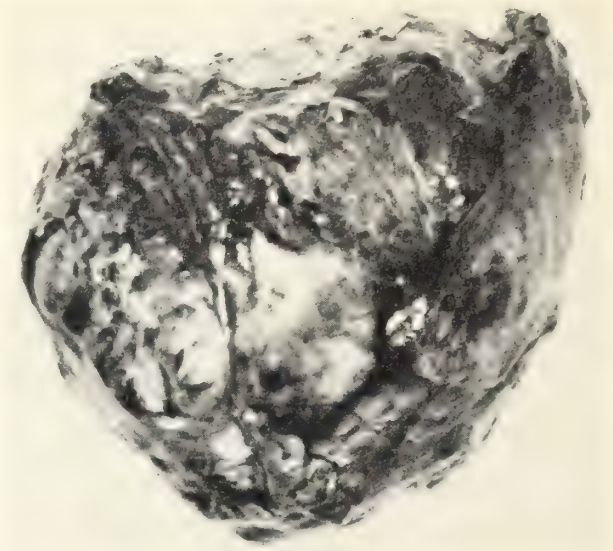

Fig. 2. The pheochromocytoma excised one month following coronary artery bypass.

with phenoxybenzamine, but recently, alpha-methyl paratyrosine, a synthesis inhibitor of catecholamines, has become available. Very effective in chronic management of patients with unresectable pheochromocytoma, alpha-methyl paratyrosine has also been shown to be safe and useful in preparation of patients for removal of pheochromocytoma.

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ABSTRACTS

These abstracts were selected from those presented at the annual meeting of the Virginia Society of Hematology on June 11 and 12, 1982, at Virginia Beach.

A Statistical Study of Drug Effects on Routine CBCs at Western State Hospital. Karl F. Menk, MD, Dorothy Evans, MT, and Lewis Cardwell, *Staunton.*

Many changes, *e.g.*, leukocytosis, have been found in the routine CBCs of some Western State Hospital patients. Many of these patients are being treated with lithium, phenytoin, carbamazepine, phenobarbital and valproic acid.

In an attempt to study drug-related changes in the CBCs of patients, the CBC values of all patients on medication for three consecutive months were entered into a computer and frequency curves with normal, SD ± 1, and SD ± 2 values were calculated. These values were entered into a grid composed on the ordinate of the CBC items and composed on the abscissa of the above drugs.

When changes in the grid showed abnormal items in the CBC of unusual frequency, individual patients' charts were pulled and studied. Possible mechanisms for the changes will be briefly discussed.

Systemic Mastocytosis: Case Report. Georgean G. deBlois, MD, Frederick G. Behm, MD, and William T. Dabney III, MD, *Richmond.*

A 58-year-old white male presented for a workup of reflux esophagitis and stricture in January 1980. At this time thrombocytopenia was discovered, and a bone marrow biopsy revealed minimal areas of fibrosis. Later, splenomegaly developed, the thrombocytopenia persisted, and the patient underwent repeat bone marrow biopsy and aspirate in October 1981. During the procedure, the patient became severely flushed and tachycardic. The biopsy revealed numerous granulomatoid lesions throughout the bone marrow. The aspirate showed normal hematopoiesis and increased numbers of mast cells. A diagnosis of systemic mastocytosis was made.

Mastocytosis is associated with five clinical syndromes: urticaria pigmentosa without systemic involvement; skin lesions with bone change; skin lesions with systemic involvement; mastocytosis and leukemia; systemic mastocytosis without urticaria pigmentosa. The last syndrome is rare and presents a diagnostic challenge. These clinical entities as well as basic mast cell physiology will be reviewed.

Ascending Myeloencephalopathy Due to Intrathecal Vincristine Sulfate: A Fatal Chemotherapeutic Error. A. N. Walker, MD, M. E. Williams, MD, J. P. Bracikowski, MD, and J. T. Carpenter, MD, *Charlottesville.*

Fatal myeloencephalopathy resulted from accidental intrathecal administration of vincristine sulfate (VCR) in a 16-year-old male undergoing maintenance chemotherapy for acute lymphocytic leukemia. The patient developed a progressively ascending chemical meningoencephalitis, leading to deep coma and loss of brain stem function by seven

days and death 36 days after injection. Multiple samples of cerebrospinal fluid (CSF) and serum were assayed for VCR; CSF levels of VCR were at least 200-fold higher than serum levels. At autopsy, all regions of the brain and spinal cord that had been in direct contact with the CSF were necrotic. Microscopically there was total neuronal loss with necrosis and gliosis in the affected regions. Conspicuous in these areas were gemistocytic astrocytes, ependymal cells, macrophages, and vascular endothelial cells that had undergone mitotic arrest and chromosomal breakage, presumably due to a direct effect of VCR on the spindle apparatus of the dividing cells.

There have been three previously reported cases of accidental intrathecal VCR administration. All three patients experienced similar ascending meningoencephalitis and death three to 14 days after injection. Attempts to exchange the CSF with saline with or without hydrocortisone had no effect on the clinical course, even when performed immediately after injection of VCR.

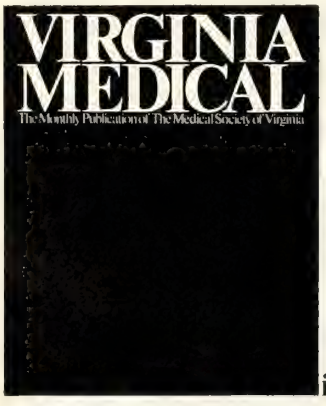
No treatment for this devastating iatrogenic error exists, underscoring the importance of preventive measures in chemotherapy administration.

Ophthalmic Lymphoid Neoplasm: Case Report and Review. Cyrus Makoui, MD, and Shakur Hamidi-Toosi, MD, *Salem.*

Orbit and occular adenexa lack lymphoid tissue, and any lymphoid tumor involving these areas is considered as extranodual neoplasm. Conjunctival tissue, on the other hand, contains small amounts of lymphoid tissue with some follicular arrangement. These anatomical features explain the differences in biological behavior between lymphoid tumors of the orbit, which mainly are malignant lymphoma, and conjunctival lesions, which primarily represent hyperplasia of preexisting lymphoid tissue. No significant clinical differences in terms of sex, age and presenting symptoms and signs between benign and malignant groups are present.

Based on histological criteria, ophthalmic lymphoid neoplasm is divided into inflammatory pseudo tumor, pseudo lymphoma, atypical reactive hyperplasia and malignant lymphoma. Approximately 17% of conjunctival lesions and 50% of orbital lesions are malignant. In the malignant group, 75% ultimately will manifest systemic disease; however, the remaining 25% will not disseminate. Although use of one micron thick section and electron microscopy has enhanced morphological identification, still morphological identification of benign and malignant groups is difficult and, in many instances, a great deal of uncertainty exists. By utilization of modern histochemical and immunological techniques and identification of B and T cell subpopulation, orbital lymphoid tumors can be divided into two major categories: 1) polyclonal group composed primarily of T cells mixed with several subtype of B cells and 2) monoclonal group primarily composed of B cells usually of one subtype mixed with smaller number of reactive T cells. Almost all occular lymphomas are non-Hodgkin lymphoma of B cell type and T cell lymphomas has yet to be reported.

A 68-year-old white male recently was treated for pseudo lymphoma of the right orbit at the Salem Veterans Administration Medical Center. The patient presented with 4 mm proptosis of right eye and a palpable firm mass at the superior nasal corner of the right orbit pushing the eye slightly down and outward. Biopsy of the lesion was interpreted as pseudo lymphoma. No treatment was given. One and one-half years later, the patient returned with 7 mm exophthalmus and diplopia. The mass was larger, extending over the temporal side of the globe 4-5 mm away from the corneo-scleral limbus. Systemic workup for malignant lymphoma and thyroid disease was negative and biopsy of the lesion was again interpreted as pseudo lymphoma. Six weeks of therapy with high dose prednisone did not result in any significant improvement. The patient was treated with a total of 1980 rads external beam radiation with dramatic clinical response. He remained in remission and is currently asymptomatic at six months post-radiation therapy.



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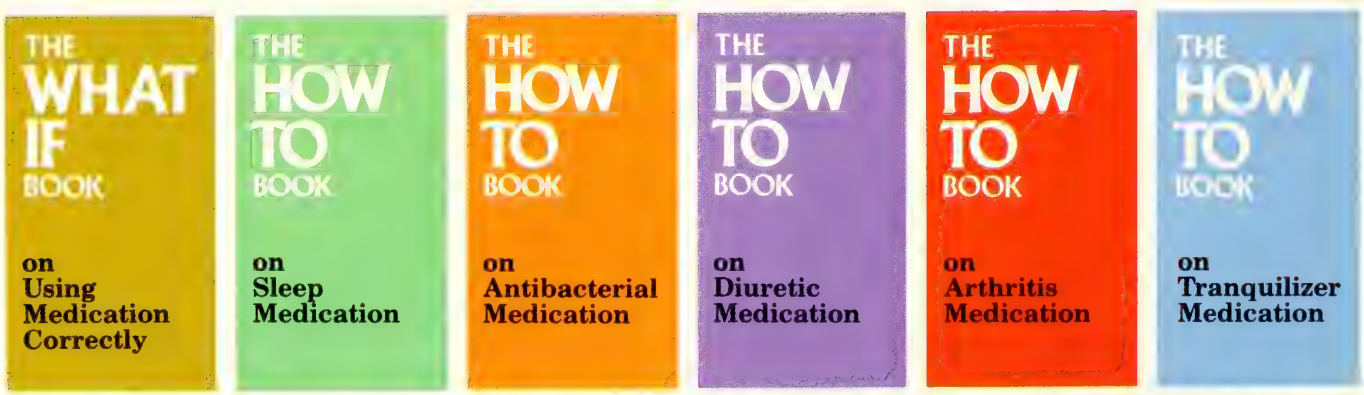

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Dr. Robert K. Jarvik with a model of the artificial heart. *Wide World Photos*

VIRGINIA MEDICAL

EDITORIAL

Barney Clark: "Odysseus, off on a last adventure"

His quick smile blossomed on the front pages of the papers. Television and radio trumpeted his name and title: Barney Clark, first recipient of a permanent artificial heart. In television's color, the studio shot was of a man with the bloom of health on his cheek. We knew the lie. Progressive heart failure had repeatedly throttled him to the point of cyanosis and desperation; his eyes were dark and hollow from ICU sleeplessness.

What would Kübler-Ross make of this man, clearly dying? He did not go to Salt Lake City to make his peace, though he knew his days were numbered; the record of the calves that had carried the plastic, pneumatic-driven pump told him that. He went in order to live on his own terms, not yet ready for the big sleep. Odysseus, off on a last adventure, unable to die calmly in his hard-won home. No matter the pain, the little humiliations, the uncertainty—he would not give in.

What are we to make of this man, squandering resources in a quixotic search for more tomorrows? The meter for such care must run at something like $100/hour, $2,400/day, $72,000/month. Had he not been bombarded, as have we all, with news of deficits, medical costs as percentage of gross national product, need to cut back? The equation is simple: number of those demanding service times cost of service equals cost of equipment and bedspace plus salaries.

The population at risk is largely older: 11% requiring 20% of the cost of care. The "graying of America" is established. The group is growing and will continue to grow. Longer-lived, healthier and wealthier than any predecessors, older Americans are fed daily information about research and developments. As with their younger counterparts, they have few qualms about the hot new treatments, as long as dignity can be preserved. Under the British National Health Service, dialysis is refused to all over 55. Here, the elderly continue to demand increased medical services, both personally and through the political process. The octagenarian with a pacemaker about to receive a hip replacement causes no raised eyebrows.

Restricting the availability of newer diagnostic and treatment modalities as a means to controlling costs was a failure of the '70s, though a few diehards fight on. Economies of scale could reduce somewhat the fee/case/doctor by exploiting use of the MD's time, but that approach was aborted in the '60s with brisk expansion of the nation's medical schools. This year's .5% reduction in the first-year medical school population will not be felt for at least five years. Economies of scale are not possible for the other hands-on people without compromising quality.

Only one conclusion remains: medical costs must go up. Wall Street recognizes this in paying a

handsome multiple for any health-related stock. The market is predicting exactly the opposite of what we all profess to wish: decline or stabilization of medical costs as a percentage of GNP. Purchasers of these stocks expect to be rewarded in the future with accelerating earnings.

Just as the world of medicine today differs so drastically from that of only ten years ago, so the future will be different from what we know now. Myoelectric replacement limbs and implantable, multi-program, dose-delivery systems—even the non-initiated sense that we stand on the verge of a technological explosion. The desirable becomes the possible becomes the necessary even as we watch. Only the untidiness of the process offends us.

There have been others like Barney Clark, though the publicity surrounding them was less rabid, and there will be others in the future. The first hemodialysis patient suffered almost every conceivable complication of chronic renal failure, thereby training a generation of nephrologists.

May Barney Clark's odyssey be similarly fruitful.

HARRY W. EASTERLY III, MD

"Why do doctors complain about Medicare?"

NO DOUBT you have heard as often as I have the question, "Why do doctors complain so much about Medicare?", usually followed by, "They are all getting rich from it, aren't they?"

To answer that doctors as an aggregate are not in medicine for money is met by an "Uh huh". The questioner, you see, has read about Medicare fraud or he has watched "60 Minutes" or both. To relate Medicare to socialism is equally fruitless. The questioner doesn't want to hear that this country was built on individual initiative; that government was established by the people and for the people; that the only power a government has or should have is the authority granted by the people under its Constitution or by amendments thereto. He doesn't want to hear that socialization of yet another segment of society leads us ever closer to a totally socialized country with governmental control over every aspect of our life and the inevitable loss of freedom which follows.

Woodrow Wilson said, "Liberty has never come from government . . . the history of liberty is the history of the limitation of governmental power, not the increase of it."

The questioner doesn't see that loss of liberty leads to strangulation of initiative and the inevitable drop in production. He doesn't know or care that an estimated one-third of the cost of health care is attributable to governmental control. He doesn't want to relate this to the effect growth in government has historically had on reduction of the gross national product of this and other nations.

Excerpted from the inaugural address of the president of the Richmond Academy of Medicine, which was presented on January 11 in Richmond.

Sir Winston Churchill said it best in 1945 when he was ousted from office by the Socialists:

I do not believe in the power of the state to plan and enforce. No matter how numerous are the committees they set up or the ever-growing hordes of officials they employ or the severity of the punishments they inflict or threaten, they can't approach the high level of internal production achieved under free enterprise. It is this vital creative impulse that I deeply fear the doctrines and policies of socialist government have destroyed. Set the people free—get out of the way and let them make the best of themselves . . . I am sure that this policy of equalizing misery and organizing scarcity instead of allowing diligence, self-interest and ingenuity to produce abundance has only to be prolonged to kill this British island stone dead.

What a prophet! In 1976, 31 years later, 60% of Britain's GNP was consumed by the government (while the US government confiscated 22.8% of our GNP), and those in England who earned over $40,000 were taxed at 83%.

The person who asked the question regarding doctors' complaints and Medicare doesn't really want an answer. He already has one. To him, this has nothing to do with democracy. Health care costs too much and doctors are responsible; they must be controlled.

MEDICARE was designed for the entire nation. The only qualification was age 65. As with Social Security, there have been expensive additions—chronic dialysis, for example. When govern-

ment expenditures began to grow, the cry went out for more control. Several Medicare frauds were publicized. The names of all doctors and groups of doctors earning more than $100,000 per year from Medicare were published in local newspapers across the land implying impropriety. With public opinion at a fever pitch and physicians intimidated, the most elaborate system of controls ever devised for any social program was unveiled—health systems agencies for control of everything medicine builds or buys and PSROs for everything we do. Out of Washington poured the "numerous committees" and "hordes of officials".

Were PSROs and HSAs effective in controlling the cost and maintaining the quality of medical care? The Office of Management and Budget studied the situation carefully and found both ineffective. Its recommendation: Phase out both programs. And what did this aspect of "free" medical care cost the public? No one really knows, but it would be a safe bet that the money spent on HSAs and PSROs would have paid for medical care for the truly indigent well past the year 2000.

And who really benefits from these programs? As with every other social program, the primary beneficiaries are the government planners, enforcers and administering employees. It is because of such elaborate programs for control that very few of the dollars allocated ever find their way to the intended use.

How much does government inflate the cost of medical care? When you consider that about one-third of the hospital bed charge is spent complying with federal regulations, that Medicare pays only 80%, that hospital charges to private pay patients must be inflated to make up the difference, and that the average certificate-of-need application costs $25,000—God only knows!

You begin to think that if government were removed from medicine, we could easily afford the advances in technology and we could realistically expect to conquer heart disease, cancer and other dread diseases. If, on the other hand, government is allowed its continued growth in medicine, costs will continue to rise and the result will be a product we cannot afford.

Until we and others face squarely the real issue in containing health care costs, true success is impossible. The number one reason why health care costs have risen faster than all other goods and services, except energy and government, is the government itself. The second reason is malpractice litigation. The people will decide whether they want more or less government in medicine and unfortunately that decision will not be made intelligently, all they need is a promise of "free" medical care. The fact that they will pay far more comes later, and by then it is difficult to understand that the actual cause of this rise in price is the layer after layer of bureaucracy of governmental control.

What can we do? We must acknowledge that the cost of medical care is too high and in our individual practices we must double our efforts to contain health care costs by

- being aware of costs of procedures, tests and prescribed treatment;
- providing health care on an outpatient basis whenever possible;
- consider adjusting fees downward when patients have limited funds.

We must also get involved in community activities and participate in the political process. We must reassume the leadership role in society that lately we have neglected. The responsible voice of the physician needs to be heard now as never before.

In the past six years, the national debt has increased from $632 billion to over $1 trillion. A significant part of this increase is due to government's attempt to provide and to regulate medical care. We need to point out that the cost of medicine to a large degree is the price we pay for socialism . . . that stripped of socialism the cost of health care would certainly fall in line with other goods and services—for example, a recently purchased heating blanket for the recovery room which met all regulations cost $1,300; the job could have been done just as safely by one that can be purchased at a department store for $15 . . . that stripped of socialism, medicine would be able to devote more time, effort and money toward advancing cost effective technology . . . that what is happening to medicine under bureaucratic control is a reflection of what is happening to the country as a whole.

The man who asked, "Why do doctors complain so much about Medicare," needs to be answered. I hope you will help me with that task.

Ronald K. Davis, MD

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Richmond VA 23226

HISTORICAL NOTES

President Andrew Jackson: Despite Infirmities, a Strong Leader

While many United States Presidents have suffered from significant illnesses while in office, our seventh President, Andrew Jackson, who served two terms in office from 1829 to 1837, seems to have been troubled with more major illnesses and injuries over a longer period of time than any President from Washington to Reagan.

"Old Hickory" was born in 1767 in Waxhaw, North Carolina. When he was 14 years old, he and his brother, Robert, were captured by British soldiers. While in prison, Andrew refused to shine a British's officer's boots and was cut by a saber on his face and one wrist. The scars of these injuries remained with him throughout his life.

Both Andrew and his brother contracted smallpox during this imprisonment and escaped during the course of the disease. Robert died, but Andrew survived, walking 40 miles while having chills and fever, but he was sick for four months thereafter.

In 1806, Jackson fought a duel with Charles Dickenson, who had made insinuations about Jackson's marriage. Dickenson was killed, and Jackson was severely wounded by a bullet in his chest that narrowly missed his heart. A pulmonary abscess and chronic bronchial infection developed from which he never fully recovered. Chills and fever, cough, dyspnea, chest pain and hemoptysis recurred at intervals until his death.

In 1813, Jackson organized and led more than a thousand volunteers in battle against the Creek Indians, which resulted in a massacre of the Indians. During this campaign he developed a troublesome diarrhea, in one day dismounting over 20 times to empty his bowels. This diarrhea became chronic; he suffered from intractable, recurrent episodes for the remainder of his life. For the acute exacerbations, he self-administered calomel; Matchless Sanitive, a proprietary drug containing 30% alcohol; and sugar of lead, which he used both orally and per rectum.

In a brawl in 1825, General Jackson was shot in the left shoulder; from the wound he developed chronic osteomyelitis with a permanently draining sinus tract. The bullet was not removed until 19 years later.

A great believer in bleeding, Jackson frequently bled himself. When, in 1828, his wife died of what was probably a myocardial infarction, he insisted that the corpse be bled from the wrist and temple.

In March of 1829, when Jackson was 62 years old, he was inaugurated as President of the United States after defeating Henry Clay in a hotly contested election. At the time of his inauguration, a report in the *Boston Medical School Journal* related: "He is a tottering scarecrow in deadly agony . . . he was hidden away and handled like fine china so that he might be well enough to take office."

In the summer of that year Jackson's dyspnea worsened and he developed edema of the feet and legs.

Jackson had disabling headaches for years, and they often confined him. They were aggravated by a blow to his head by a naval officer during the debates in the so-called "Bank War." This was the controversy in which Jackson, who wanted to break up the United States Bank, defeated a powerful coalition led by Clay, Nicholas Biddle, Daniel Webster and John C. Calhoun. Often associated with Jackson's headaches were nervous aberrations, rage and insomnia. They never prevented him, however, from continuing with his old habits of smoking a pipe, chewing tobacco, and drinking gin.

In spite of fragile health, Jackson was elected to a second presidential term in 1833, but shortly thereafter, during a trip to New England, he collapsed in Portland, Maine, as a result of a massive pulmonary hemorrhage. After emergency treatment, he was transported by steamboat to Washington. Later he recuperated in Hampton Roads, Virginia, at the newly built Fort Wool, the island fortress that Virginians nicknamed "Rip-Raps."

Returning to the White House, he was soon confined to his bed because of an exacerbation of the osteomyelitis, together with severe abdominal distress. From Charlottesville as consultant came Thomas Jefferson's trusted physician, Dr. Robley

Dunglison; his greatest service was to insist that the President not bleed himself further. Jackson later consulted the famous Dr. Philip Syng Physick of Philadelphia, known to us today as the "father of American surgery", but he emphatically told Doctor Physick that he could not refrain from smoking or from drinking coffee and alcohol.

The edema progressed and involved the President's face. He was blind in one eye, with failing vision in the other. The diarrhea worsened. The chest pain and dyspnea increased. Throughout, Jackson self-administered calomel and sugar of lead.

During his last days in the White House, President Jackson had another pulmonary hemorrhage and was thought to be dying. At the conclusion of his farewell address he stated, "My race is nearly run . . . before long I must pass beyond the reach of human events."

In fact, he lived eight more years, in his home in Tennessee, though as an enfeebled semi-invalid with steadily deteriorating health. His last public appearance was in New Orleans in 1840, and he died at the age of 78 on June 8, 1845.

The most likely cause of Jackson's death was nephrosis caused by a generalized amyloidosis resulting from long-standing, recurrent infections, pulmonary abscess, osteomyelitis, and extensive inflammatory bowel disease (amebiasis or idiopathic ulcerative colitis). All of these were more or less active for over 40 years. In addition, his chronic consumption of mercury and lead products, together with frequent venesection, aggravated the basic pathological states.

To what degree, if any, did Jackson's illnesses and disabilities diminish his effectiveness as the leader of his country? One can only speculate. He was a powerful President, with courage and will. Even when criticially ill, he continued to exercise strong leadership.

CHARLES M. CARAVATI, MD

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VIRGINIA MEDICAL OBITUARY

James B. Funkhouser, MD

Dr. James Bauer Funkhouser, Richmond, who was instrumental in the development of Virginia's system of mental hygiene clinics, died January 24 in Richmond after a long illness. He was 69 years old.

Born in Indianapolis, Dr. Funkhouser was educated at Indiana University and its medical school and interned at the Marion County, Indiana, General Hospital. He became an associate clinical professor of psychiatry at the Medical College of Virginia and, following World War II, chief of psychiatry at McGuire Veterans Administration Hospital. Well-known for his proficiency in the reading of electroenceophalograms, he was certified by the American Board of Electroencephalography.

In 1957 Dr. Funkhouser joined the state government and served as acting superintendent of Eastern, Central, Western and Southwestern State Hospitals. At the time of his retirement in 1979 he was deputy commissioner of the State Department of Mental Health and Mental Retardation and a member of two of the Governor's committees, Hospital Advisory and Overall Advisory on the Needs of Handicapped Children, and of the Virginia Council for the Deaf.

A past president of the Neuropsychiatric Society of the Virginias, Dr. Funkhouser had been a member of The Medical Society of Virginia for 43 years. He belonged also to the Richmond Academy of Medicine, the Group for the Advancement of Psychiatry, American Psychiatric Association, American Academy of Neurology, and the Association for Research of Nervous and Mental Disease.

A son, Dr. John J. Funkhouser, practices medicine in West Falmouth, Massachusetts.

J. W. Sinclair, MD

Dr. James Ward Sinclair, longtime Warrenton surgeon, died January 5 at his home at the age of 87. He had celebrated his 50th wedding anniversary with his wife, Harriet, on January 3.

Born in Toronto, Canada, in 1895, Dr. Sinclair was graduated from the University of Toronto and its Faculty of Medicine. He served in World War I as a medical corps captain with the Canadian Expeditionary Forces in Canada and England and remained in England as surgical registrar of the Royal Southampton and Hants Hospital at Southampton. After a visit in 1926 to this country, he settled in Fauquier County.

Early in 1942, Dr. Sinclair enlisted in the Royal Canadian Navy Volunteer Reserve and became surgeon commander of the HMS Cornwallis in Nova Scotia.

Dr. Sinclair had been a member for almost 50 years of both the Fauquier County Medical Society and The Medical Society of Virginia.

James L. Davis, MD

Dr. James Lucius Davis, general practitioner in Waynesboro for almost 40 years, died January 2 at Waynesboro Community Hospital. He would have been 71 on January 19.

Born at Mt. Valley Racoonford in Orange County, Virginia, Dr. Davis was educated at Virginia Polytechnic Institute and was graduated from the University of Virginia School of Medicine in 1936. He interned at hospitals in New Jersey and New York, then practiced medicine in the West Virginia and Kentucky coal mining areas for three years before setting up his Waynesboro practice. During World War II he served with the US Army Air Corps in the African theater.

Early last year Dr. Davis was presented with the Waynesboro Exchange Club's Book of Golden Deeds Award, which recognizes those "whose selfless toil and sacrifice for public good is motivated only by an innate desire to help others."

Dr. Davis came to membership in The Medical Society of Virginia 35 years ago through the Augusta County Medical Society.

J. J. Moriarty, MD

Dr. James J. Moriarty, Alexandria, died on November 15, 1982, at the age of 76. He had conducted a general practice in Alexandria since 1946.

Educated at Loyola University and the Midwest Medical School, Kansas City, Dr. Moriarty interned at hospitals in New York State. During

World War II he was a Public Health Service officer in Virginia, serving in the Lynchburg area and at Ft. Belvoir. A member of the Alexandria Medical Society and The Medical Society of Virginia, Dr. Moriarty belonged also to the American Academy of Family Physicians.

J. B. Adams, MD

Dr. James Belt Adams, for 30 years a practicing physician in Emporia, Virginia, died December 31, 1982, in a Richmond hospital. He was 60 years old.

Born in Halifax County, Virginia, Dr. Adams was graduated from the University of Richmond and the Medical College of Virginia. He interned at Johnston-Willis Hospital, Richmond, then went to Emporia to establish a practice with his late wife, Dr. Avis Branch Adams, for whom he named the facility he founded in 1971, the Avis Branch Adams Christian Convalescent Center.

Dr. Adams had been chief of staff of Greensville Memorial Hospital and at the time of his death he was the Greensville County medical examiner. He belonged to the Southside Virginia Medical Society, The Medical Society of Virginia, and the American Academy of Family Physicians.

Charles W. Scott, MD

Dr. Charles William Scott, Farmville, former superintendent of Piedmont State Hospital, was killed in a motor car collision in Farmville on January 13. He was 83.

Dr. Scott was graduated from the University of Virginia School of Medicine in 1923. He received training at the University of Virginia Hospital and Blue Ridge Sanatorium, Charlottesville. Soon after moving to Farmville in 1924, Dr. Scott began conducting diagnostic clinics for the state Health Department. In 1942 he was named superintendent of Piedmont State Hospital, Burkeville, remaining in that post for 26 years. Following his retirement in 1968, he continued to conduct a weekly chest clinic for the Prince Edward Health Department, serving also as resident physician for the Holly Manor Nursing Home.

In 1960 the Virginia Tuberculosis Association gave Dr. Scott its Douglas Southall Freeman Award.

A member of the American College of Chest Physicians and the American Thoracic Society, Dr. Scott was also a long-time member of The Medical Society of Virginia and the Southside Virginia Medical Society.

Memoir of Snowden C. Hall, Jr. 1903-1982

By Clifford G. Gaddy, MD and Walter Fitzgerald, MD

We wish to pay tribute to our colleague and friend, Dr. Snowden Cowman Hall, Jr., who died on April 17, 1982, after more than 46 years of devoted service as a revered physician to the people of Danville.

Dr. Hall was born November 25, 1903, in Northumberland County. He graduated Phi Beta Kappa from the College of William and Mary and subsequently taught school and became a principal in Northumberland County. Later, he attended Harvard Medical School and graduated in 1930. He did his internship at Boston City Hospital and his residency at Duke University Hospital. He taught at the University of Alabama Medical School prior to coming to Danville in 1936. During World War II, Dr. Hall served as a Commander in the United States Navy.

In 1937 he married the former Ida Shankle Hardman, who survives along with two physician sons, Dr. Snowden C. Hall III of Richmond and Dr. James H. Hall, Mount Vernon, Maine, and one daughter, Ida C. Hall, of Kilmarnock.

Although Dr. Hall retired from private practice in December, 1975, he continued to serve as a consulting physician at the Southern Virginia Mental Institute in Danville and also continued his regular attendance at hospital staff meetings and scientific programs.

Dr. Hall was a member of Mount Vernon United Methodist Church and served on its administration board and board of trustees. He was a past chairman of the State Board of Medical Examiners.

A quiet and unassuming man, Dr. Hall was noted for his excellence in all he undertook. He loved quail hunting and was a great competitor in golf.

He was held in highest regard by all of us who knew him and his presence among us will be greatly missed.

Prepared and published at the request of the Danville-Pittsylvania Academy of Medicine.

WHO'S WHO

Two retired Waynesboro physicians, **Dr. David Tyler** and **Dr. I. Earl Holmes,** were honored at a banquet given late last year by the medical staff of Waynesboro Community Hospital. Both doctors received plaques of appreciation to commemorate the occasion.

Dr. Tyler's plaque, presented by **Dr. John R. Talbert,** commended him for "24 years of outstanding medical care in obstetrics and gynecology." A native of Richmond, Dr. Tyler was graduated from the Medical College of Virginia. He started practicing in Waynesboro in 1956 and retired two years ago for reasons of health.

Dr. Holmes' citation, presented by **Dr. James E. Nathe,** recognized "25 years devoted to the uncompromising care of radiology patients." He was the first permanent radiologist at Waynesboro Community Hospital and headed that department until his retirement in 1982. A native of Missouri, he was graduated from George Washington University School of Medicine and began his Waynesboro practice in 1957 after several years of practice in nearby Staunton.

At the same meeting, **Dr. Ronald W. Rau** was elected president of the hospital's medical staff.

Dr. David E. Longnecker was named Distinguished Professor of Anesthesiology by the University of Virginia's board of visitors at its recent quarterly meeting. Dr. Longnecker came to Charlottesville in 1974 from the University of Missouri.

At its annual workshop in Fishersville recently, the Virginia Rehabilitation Association honored **Dr. Reno Porter,** Richmond, with its Roy M. Hoover Award for his service to Virginians with disabilities. Dr. Porter, a cardiologist, is chief medical consultant for the Virginia Department of Rehabilitative Services and is also on the faculty of the Medical College of Virginia.

Almost 2 million coins in the Luray Caverns' "wishing well" were fished out recently, totted up to $48,436, and presented to **Dr. Nuzhet O. Atuk,** University of Virginia kidney specialist, for his research into the therapeutic application of calcium entry blockers. The owners of the Shenandoah Valley tourist attraction collect the coins about every five years and give the money to medical or charitable organizations.

Dr. Robert A. Woolfitt, Norfolk, has been named chairman of the Department of Radiology at Eastern Virginia Medical School. A 1970 graduate of the West Virginia University School of Medicine, Dr. Woolfitt joined EVMS in 1978.

When the Merchants Association of Roanoke Valley named four Fathers of the Year recently, the one honored for his contributions to family life was **Dr. Robert E. Paine, Jr.,** Roanoke. Dr. Paine left private practice in 1967 to work with alcoholics, and, although now retired, he continues as a volunteer at the VA's Alcohol Rehabilitation Center in Salem. He has been president of the Roanoke Valley Heart Association and in 1966 was medical director of the Virginia State Association of Rescue Squads.

At the recent annual meeting of the American Academy of Facial Plastic and Reconstructive Surgery, **Dr. Robert W. Cantrell,** Charlottesville, was reelected vice president for the Southern Region. Dr. Cantrell is chairman of the Department of Otolaryngology and Maxillofacial Surgery at the University of Virginia.

In Stuarts Draft, **Dr. John W. Forbes III** was drafted as local chairman of a campaign for funds and "bike-a-thon" for the Virginia Chapter of the Cystic Fibrosis Foundation.

Newly elected president of the American Heart Association, Virginia Affiliate, is **Dr. Michael A. Moore,** nephologist who practices in Danville. Named at the same time to serve as president-elect was **Dr. Hermes A. Kontos,** Richmond cardiologist.

Dr. Randolph M. Jackson, Winchester, was reelected assistant secretary of the American Society of Anesthesiologists at its recent meeting in Chicago. Dr. Jackson is a past president of the Virginia Society of Anesthesiologists.

The board of directors for the Virginia Easter Seal Society selected **Dr. Nancy M. Welch,** Roanoke, as 1982 Volunteer of the Year. Dr. Welch is director of the Alleghany Health Department and since 1975 has been a physician at Camp Easter Seal in New Castle, Virginia.

At the American Academy of Otolaryngology's annual meeting in New Orleans, **Dr. Gary L. Schechter,** Norfolk, received an Honors Award for his contributions to the academy's educational programs.

These nine Medical Society of Virginia members were elected to fellowship in the American College

of Chest Physicians in 1982: **Dr. Hormoz Azar**, Norfolk; **Dr. Michael Z. Blumberg**, Richmond; **Dr. John B. Cleary** and **Dr. Edward A. Lefrak**, Fairfax; **Dr. Bolling J. Feild**, Petersburg; **Dr. Philip B. Fuller**, Fredericksburg; **Dr. James A. Long**, Waynesboro; **Dr. Thomas P. Splan**, Newport News; and **Dr. Roger B. Wigton**, Burke.

To **Dr. Leo J. Dunn,** Richmond, will go the annual award of the Louise Obici Memorial Hospital, Suffolk. The award is given for "outstanding contributions to the medical profession."

Chairman of the Department of Obstetrics and Gynecology at the Medical College of Virginia, Dr. Dunn recently was appointed president of the American Board of Ob-Gyn. He has been president of the Foundation of the American Association of Obstetricians and Gynecologists; chairman of the Council of Resident Education in Ob-Gyn; and a member of the AMA's Residency Review Committee. He headed The Medical Society of Virginia's Maternal Health Committee from 1975 to 1980.

The presentation is to be made at the hospital's annual clinical conference on April 6 at the Suffolk Armory.

Many of The Medical Society of Virginia's component societies elect officers at the start of the year; here are some of these newly selected slates.

Dr. John P. McDade is the new president of the Alexandria Medical Society, and these will serve with him: **Dr. Antonio M. Longo,** president elect; **Dr. Peter B. Collis,** vice president; **Dr. A. Roger Wiederhorn,** secretary; and **Dr. John W. Klousia,** treasurer.

To serve the Richmond Academy of Medicine, these officers: **Dr. Ronald K. Davis,** president; **Dr. Wyatt S. Beazley III,** president elect; **Dr. J. Latane Ware** and **Dr. E. Randolph Trice,** vice presidents; **Dr. Lindley T. Smith**, secretary; **Dr. Edward A. Zakaib,** treasurer; and **Dr. Marcella F. Fierro,** sergeant-at-arms.

For the Augusta County Medical Society, **Dr. Donald T. Fowler** is the new president; **Dr. John P. Heatwole, Dr. Robert C. Kluge** and **Dr. J. W. Forbes** are vice presidents; **Dr. Stephen A. Howlett** is secretary; and **Dr. R. Mardre Bell** is treasurer.

New president of the Northern Virginia Medical Society is **Dr. Thomas E. Patteson III,** Front Royal. **Dr. Terry Sinclair,** Winchester, is president elect, and **Dr. Charles H. Miller** is secretary-treasurer.

Officers of the Halifax County Medical Society for 1983 are **Dr. Gurnam S. Bajwa,** president; **Dr. William M. McConahey III,** vice president; and **Dr. Rufus H. Gordon,** secretary-treasurer.

Leading the Fairfax County Medical Society in 1983 are **Dr. Paul G. Rochmis,** president; **Dr. William L. Rich III,** president elect; **Dr. Louis Q. Pugsley,** vice president; **Dr. Delosa A. Young,** secretary; and **Dr. Edgar H. Soifer,** treasurer.

New President of the Southwestern Virginia Medical Society is **Dr. John Boniface, Jr.,** Galax. Serving with him as secretary is **Dr. Donald B. Nolan,** Roanoke.

The Fredericksburg Area Medical Society has elected these new officers: **Dr. Louis B. Massad,** president; **Dr. Stewart E. Kohler,** president elect; and **Dr. Leroy J. Essig,** secretary-treasurer.

The Arlington County Medical Society has elected these officers: **Dr. Raymond R. Hoare,** president; **Dr. Robert G. Bullock,** president-elect; **Dr. Roger D. Cornell,** vice president; **Dr. Frank A. Pettrone,** secretary; and **Dr. William L. Stone III,** treasurer.

Double-barreled salute: from left, Dr. Earl Holmes, Dr. David Tyler, Dr. Ronald Rau.
Ed Berlin photograph courtesy the Waynesboro News-Virginian

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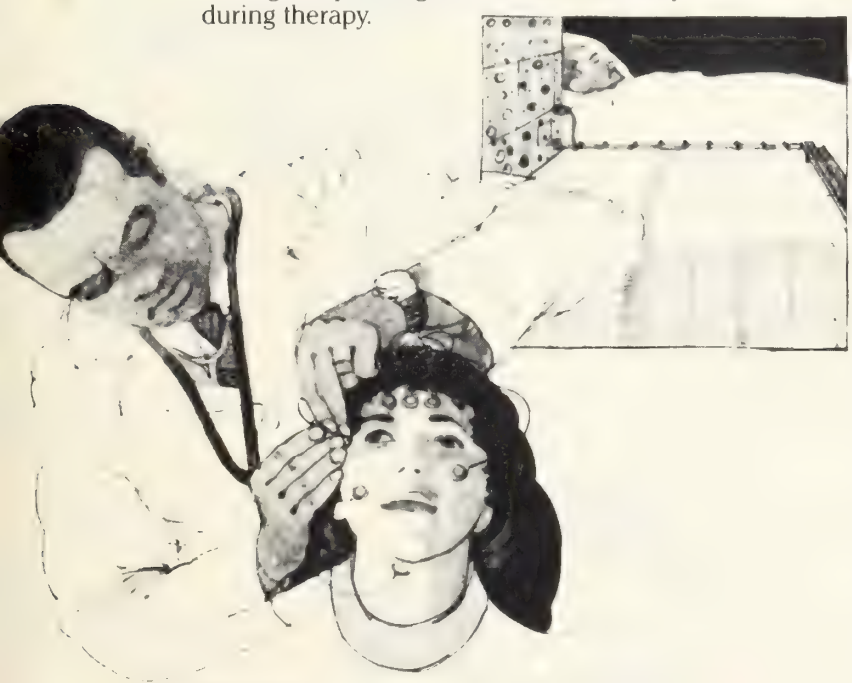

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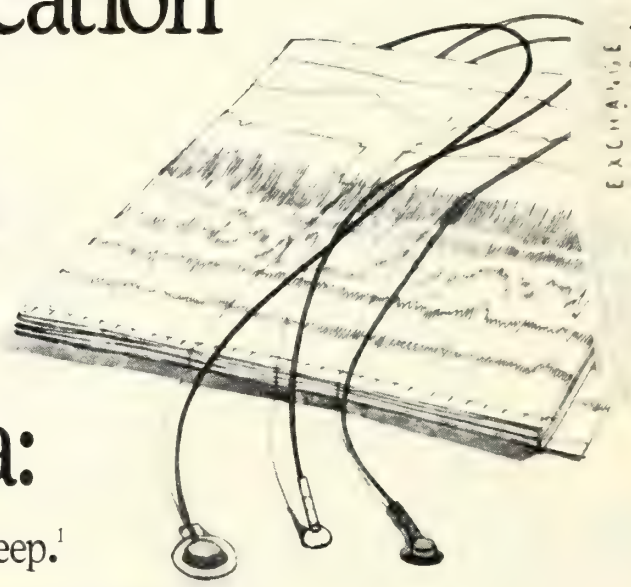

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New Physician To The Area

The Physician Recruitment Committee of Russell C... announc... ed word... Practice... setting...

New Orthopedist

Area to get new doctor

Even though the ... weeks, Dr. Robert ... were the guests of ... Saturday in Albert ... people

Dr. James S. McGinn, formerly of Prince William County, Va., has ... to Mar... his Internship, and Residency in Surgery at the United States Naval Hospital, Portsmouth, Va. He ... a member of the ... Navy from ... prior to coming to ... tinsville h... Surgery a... William Hc... sas. Va. Certified by ... Board of Su...

Ophthalmologist Opens Office

Dr. Ripon W. LaRoche II of Staun... ...kamined his first patients in ...gton on Monday.

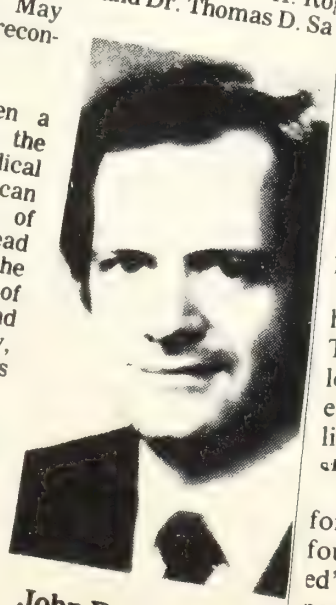
Roche, an ophthalmologist ... ...tices here in the office of Th... ... in Deavers Alley o...

Doctor joins Lakeview staff

...itten several ... in the ...c literature, ...resenting a ...e annual ...e Virginia ... of ... and this May ... recon... es.

Dr. Lyle's office w... in Lakeview Clinic.

Also on the staff ... clinic are Dr. D. B. ... coran, Dr. V. B. ... surgeons; Dr. Georg... Cornell, Dr. W. H. Ro... and Dr. Thomas D. Sa...



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..., a native of Zan... ...zania, attended ... and began his ...ies in Kampala, ...use his

Emporia has new cardiologist

By ED CONNER
I-M Staff Writer

Dr. George Eapen, a cardiologist ... from ... pract... Eap... his ... Triva... lege in... ed to ... like... small t... After ... for sev... found that "nobody talked" there. He wanted more peace and quiet ... which takes a 24-hour cardiogram, as an example.

Eapen came to the United States in 1973, and worked at ... passed his certification in cardiology.

Eapen's wife ... campus ... furnish...

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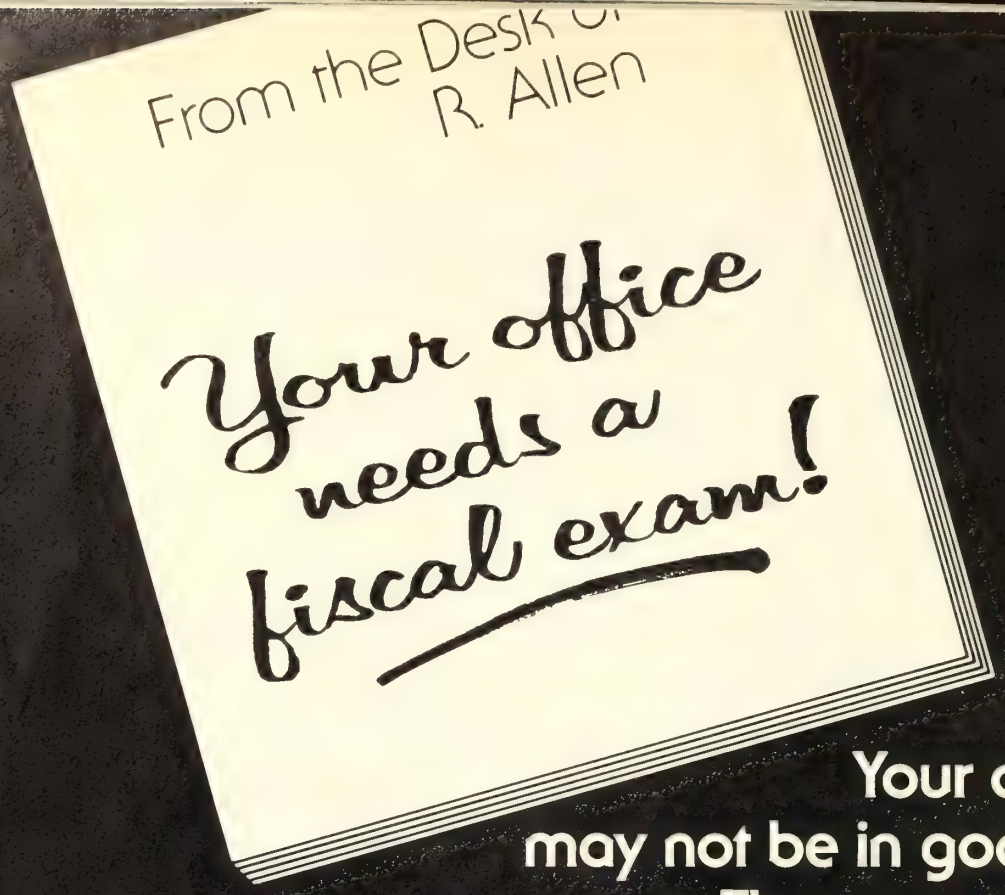

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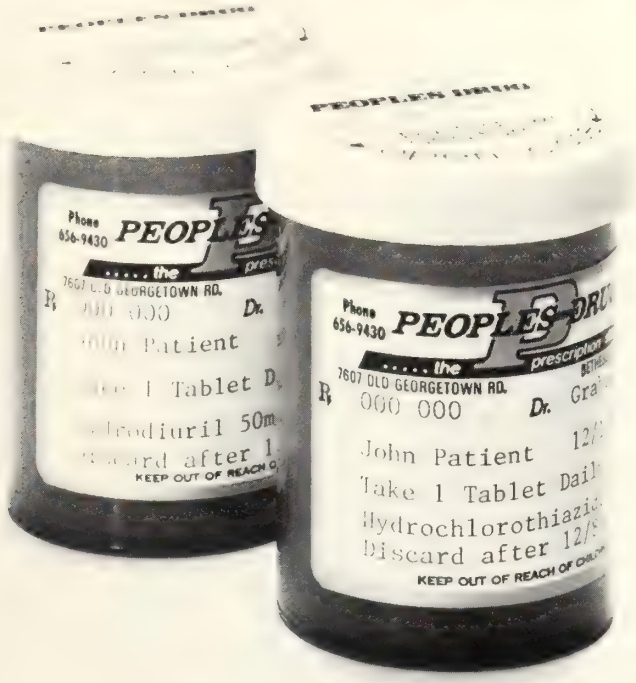

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Physician placement: There's still room in Virginia

Notwithstanding a greater output of trained physicians, there still is plenteous opportunity for setting up a medical practice in Virginia.

That word comes from a man who, as director of the Virginia Council of Health and Medical Care, has been instrumental in helping nearly 1,300 physicians locate in Virginia during the past 35 years. He is Edgar L. Fisher, Jr., a crisp and lean graduate of the College of William and Mary, who has been with the Council since 1948, two years after its founding.

Moreover, those 1,300 placements have come without a single penny of taxpayers' money but entirely through private support. Fisher is proud of that record. On expenditures last year of $265,221, he ran an organization that not only places physicians seeking to establish practice or change practice sites but also runs referral services for dentists and occupational therapists and participates in nutritional education and health careers programs. The neighboring state of North Carolina, Fisher observes, spends nearly $1 million annually in public funds to do essentially the same combination of jobs.

Dr. Wyndham B. Blanton, Jr., (left), past president of the Virginia Council on Health and Medical Care, and Edgar L. Fisher, Jr., the Council's director.

Where does the Virginia Council's money come from? From The Medical Society of Virginia, which has contributed to the Council's finances since its inception; from physicians, hospitals, industrial and banking concerns, nursing homes, foundations, trusts; and from the Virginia Hospital Association, whose contribution is earmarked for the Council's health careers program. All contributions, Fisher noted pointedly, are tax deductible.

Last year marked a slight dropoff from 1981 in the number of physicians the Council helped place in practice locations. The figures were, respectively, 38 and 46. Of the 38 placed last year, 24 were family physicians. But a year does not a trend make, contends Fisher, pointing out that the average placements since the founding of the Council has been about 35 annually.

"The numbers fluctuate from year to year; I see no particular trend," he says.

While the statistics from both the nation and Virginia show more MDs being trained,[1] "You've got to take into account," according to Fisher, "that demand is going up also, what with the availability of insurance in

various forms to most people, a growing concern for health care, and increased life expectancy."

He and his aides have produced a map of Virginia with dots showing 1,258 MDs placed through the Council from its inception 37 years ago through July 1982. The map looks as though it has come down with the measles—marked by 241 dot locations, many of them representing multiple placements.

Based on the map, Fisher was asked, weren't there now fewer localities in Virginia without an adequate number of doctors? Replying with all the modesty he could muster, he said, "Yes, largely due to what we've done."

Still, there are geographical areas in the state where family physicians are scarce. Fisher noted specifically the Southside area, stretching from Franklin in Southampton County west along the North Carolina border through Danville; the Shenandoah Valley from Strasburg south to Harrisonburg; portions of Southwest Virginia beyond Roanoke; and some of the Tidewater section and Northern Virginia, both with high populations.

But there is greater demand than supply, he pointed out, for psychiatrist, orthopedists, ear-nose-throat specialists and ob-gyns.

Fisher agrees with an article citing a gypsy streak among physicians, particularly the younger ones.[2] Doctors move more than the general populace, this report says, with the trend from the Snow Belt to the Sun Belt. Apparently they bask in patients as well as the sun in an area increasing in population. The figures cited show more than 24% of all MDs changing addresses between 1974-1978, with post-residency physicians changing professional locations at a 16% rate for the same period.

Is the Council, then, simply a bulletin board for medically needy communities to find these movable doctors?

Not at all, Fisher said. The Council prides itself on analyzing each community's request for help before trying to find a physician to fit the need. The interests of physicians already established in a community are high on the Council's priorities, along with the needs of the population to be served by the new doctor.

Two major factors are considered: 1) the number of physicians within the requested specialty practicing in the requesting area, their type of practice (private, academic, governmental), and their activity level (full-, part-time); 2) the primary population base to be served and its anticipated growth patterns.

To assess these elements, Fisher and his assistants interview established physicians, public health doctors in the area, and hospital administrators. Sometimes the field work does not support fully the expressed need, and the Council turns down the community's request.

Sometimes, too, of course, the interested physician turns down the community. Multiple factors go into that decision. One of the major ones is spouse disapproval. "You just can't overemphasize the importance of a spouse's judgment as to whether or not the physician will pick a particular location," said Fisher. "Cultural, educational, social and recreational matters rate very high with wives."

The things physicians look for when they establish practice or relocate may be changing somewhat, Fisher thinks. Recent surveys of medical school graduates indicate to him that "a trend appears to be developing wherein lifestyle, geography and other such matters are as important to these young physicians as income potential."

Fisher relies heavily for advice and guidance on an executive committee of 15 volunteer members who elect the Council's officers each year. Three physicians are serving on that committee this year: Dr. Charles E. Horton, Norfolk; Dr. Daniel N. Mohler, Charlottesville; and Dr. Ralph Ownby, Jr., Richmond.

For additional help there are the Council's trustees, among them this year these physicians: Dr. Wyndham B. Blanton, Jr., Richmond, who was president of the Council last year; Dr. M. Pinson Neal, Jr., and Dr. William T. Wilkins, both also of Richmond; Dr. Alexander D. Crosett, Kilmarnock; Dr. James B. Kenley, Virginia's Commissioner of Health; and Dr. Harold L. Williams, Newport News, President of The Medical Society of Virginia.

—CARL L. SHIRES

1. Rosinski EF: The physician "surplus": How did it happen? What to do about it? Va Med 108:628-630, 1981
2. Steiber SR: Physicians who move and why they move. JAMA 248:1490-1492, 1982

Candidates for nutritional therapy...

10,000,000 alcoholics. Ethanol may produce many effects that together bring about nutritional deficiencies, so that alcoholism affects nutrition at many levels.[1]

25,500,000 geriatric patients. The older patient may have some disorder or socio-economic problem that can undermine good nutrition.[2]

23,500,000 surgical patients. Nutritional status can be compromised by the trauma of surgery; and some operations interfere with the ingestion, digestion and absorption of food.[3]

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5,000,000 hospital patients with infections.[4] Many are anorectic and may have a markedly reduced food intake. Supplements are often provided as a prudent measure because the vitamin status of critically ill patients cannot be readily determined.[3]

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References: 1. Shaw S, Lieber CS: Nutrition and alcoholism, chap. 40, in *Modern Nutrition in Health and Disease*, edited by Goodhart RS, Shils ME. Philadelphia, Lea & Febiger, 1980, pp. 1220, 1237. 2. Watkin DM: Nutrition for the aging and the aged, chap. 28, in *Modern Nutrition in Health and Disease, op. cit.*, p. 781. 3. Shils ME, Randall HT: Diet and nutrition in the care of the surgical patient, chap. 36, in *Modern Nutrition in Health and Disease, op. cit.*, pp. 1084, 1089, 1114. 4. Dixon RE: *Ann Intern Med* 89 (Part 2): 749-753, Nov 1978 5. Committee on Dietary Allowances, National Research Council: Recommended Dietary Allowances, ed 9. Washington, National Academy of Sciences, 1980, p. 13.

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MEETINGS

April 22-23
22nd Annual Swineford Allergy Conference (University of Virginia), *Charlottesville*. 8 credit hrs., Cat. I. Fee, $50. Lee Ann Fargo, Box 225, UVa Medical Center, Charlottesville VA 22908, (804) 924-5917.

April 23
Colorectal Cancer (Chesapeake General Hospital/Eastern Virginia Medical School), *Virginia Beach*. 6 credit hrs. Fee, $40. Anita B. Theroux, Chesapeake General Hospital, PO Box 2028, Chesapeake VA 23320, (804) 547-8121.

April 22-24
Arrhythmia and Cardiac Ischemia: Diagnosis and Management (International Medical Education Corporation), *Williamsburg*. Cat. I credits. Mary A. Schroeder, IMEC, 64 Inverness Dr., E., Englewood CO 80112, toll free: (800) 525-8651.

April 22
4th Annual Spring Clinical Conference (Medical College of Virginia and John Randolph Hospital), Holiday Inn, *Chester*. Theodore C. Andrew, MD, John Randolph Hospital, Hopewell VA 23860.

April 23
Biomedical Consequences of Nuclear Weapons and Nuclear War (University of North Carolina), *Chapel Hill*. 7 Cat. I credits. Betty Neilson, Office of CME, 231 MacNider Bldg., Chapel Hill NC 27514, (919) 962-2118.

April 27-30
Annual McLemore Birdsong Conference in Pediatrics (University of Virginia), *Charlottesville*. Office of CME, Box 368, UVa Medical Center, Charlottesville VA 22908, (804) 924-5318.

April 28-30
Sexually Transmissible Diseases: An Update (Johns Hopkins University), *Baltimore*. 18 credit hrs. Fee: $250. Susan Bavaro, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

April 28-30
Emergency Medicine (Medical College of Virginia), *Williamsburg*. Fee: $195. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

April 28-30
Tutorials in Interventional Radiology and Digital Subtraction Angiography (Johns Hopkins University), *Baltimore*. Cat. I credits. Fee: $275. Carlita Kearney, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-3168.

April 29
Annual Meeting, Society for Research and Education in Primary Care Internal Medicine, *Washington DC*. J. D. Kruger, 4200 Pine St., Philadelphia PA 19104.

April 29
Pain Symposium (University of Maryland), *Baltimore*. Office of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

April 29-May 1
Clinical Management of Coronary Disease and Dual Mode Management (International Medical Education Corporation), *Arlington*. Cat. I credits. Mary A. Schrorder, IMEC, 64 Inverness Dr., E., Englewood CO 80112, toll free: (800) 525-8651.

April 29-May 1
Springfest: Program in Pediatrics (Medical College of Virginia), *Williamsburg*. Fee: $150. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

April 29-May 2
Annual Meeting, American Federation for Clinical Research, *Washington D C*. C.B. Slack, 6900 Grove Rd., Thorofare NJ 08086.

April 30
1st Annual Spring Symposium (Eastern Virginia Interhospital Medical Education Committee), *Kilmarnock*. Robert Hoyt, MD, Rappahannock General Hospital, Kilmarnock VA 22482.

May 2-4
Johns Hopkins Emergency Medicine Symposium (Johns Hopkins University), *Baltimore*. Fee $300. Diane Heydinger, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

May 4-7
Annual Meeting, Virginia Society of Ophthalmology and Otolaryngology, *Charlottesville*. Donna Strawderman, 4205 Dover Rd., Richmond VA 23221, (804) 353-2721.

May 5-7
Update in Surgery (Johns Hopkins University), *Baltimore*. 18 credit hrs. Fee, $295. Noreen Javornik, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

May 20
Frontiers of Forensic Psychiatry (University of Maryland), *Baltimore*. Michael Wessely, Education Coordinator, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

June 1-4
Review Course in Reproductive Endocrinology (Johns

Hopkins University), *Baltimore*. 37 credit hrs. Fee $375. Noreen Javornik, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

June 2-4
First Symposium on Behavioral Neurology: High Cortical Functions of the Brain (Vanderbilt University) *Nashville, Tennessee*. Mrs. Joan Sullivan, Dept. of Neurology, Vanderbilt School of Medicine, Nashville TN 37212.

June 2-3
Treating the Difficult Alcoholic Patient: Avoiding Malpractice (University of Maryland), *Baltimore*. Michael Wessely, Education Coordinator, 10 S. Pine St., Baltimore MD 21201, (301) 528–3956.

June 3-5
5th Annual Institute on Medical Hypnosis (George Washington University), *Washington DC*. Greg P. Thomas, Office of CME, 2300 K. St., NW, Washington DC 20037.

June 6-10
7th Annual Myocutaneous Flap Dissection Workshop (Plastic Surgery Specialists, Inc.), *Norfolk*. Vicky Martin, 400 W. Brambleton Ave., Norfolk VA 23510, (804) 623-1090.

June 8-12
Rehabilitation of the Brain-Injured Adult (Medical College of Virginia), *Richmond*. 22 credit hrs. Fee, $225. Sheri Rosner, Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 780-0494.

June 9-11
Advances in Pediatric Ophthalmology and Strabismus (Eastern Virginia Medical School), *Virginia Beach*. Earl Crouch, MD, EVMS, PO Box 1980, Norfolk VA 23510.

June 13-17
Advanced Practitian in Ultrasound (Johns Hopkins University), *Baltimore*. Fee, $400. Julia C. Burbridge, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

June 13-17
9th Annual Family Medicine Review Course (University of Maryland), *Baltimore*. Terry S. Young, Office of CME, 10 S. Pine St., Baltimore MD 21201.

June 19-24
Eastern Shore Medical Symposium (University of Delaware/Medical Society of Delaware), *Rehoboth Beach, Delaware*. Fee, $395. Sylvia Brocka, Division of CME, University of Delaware, 2800 Pennsylvania Ave., Wilmington DE 19806, (302) 738-8151.

June 24-26
Internal Medicine Symposium (Eastern Virginia Medical School), *Virginia Beach*. James P. Baker, MD, EVMS, PO Box 1980, Norfolk VA 23510.

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LETTERS

State Commissioner and Board of Health to Belle Fears: "We'll miss you"

The State Board of Health has presented a citation to Dr. Belle DeCormis Fears of Accomac to acknowledge her competent and faithful service to the Virginia Department of Health for the past 18 years. Dr. Fears retired on March 1. She will certainly be missed.

Dr. Fears has participated actively in local, state and national medical societies, and she has served on numerous boards and commissions, including Rural Health Systems, Mental Retardation Services Board, Area Agency on Aging, and has been a trustee for the Northampton/Accomac Memorial Hospital. She was most instrumental in the starting of the hospice movement for her district and served as its medical director.

The Board of Health and I wish her good health and happiness in her retirement.

James B. Kenley, Md,
State Health Commissioner

109 Governor Street
Richmond VA 23219

Dr. Fears surrounded by, from left, Dr. Clarence W. Taylor of the State Board of Health; Dr. William B. Crawford, Eastern Region health director; Dr. Kenley; and Dr. Fears' husband, State Senator William E. Fears.

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The office is within half a block of Stuart Circle Hospital, a 153-bed, short-term, medical/surgical hospital offering the convenience of outpatient testing for your patients. A 24-hour Outpatient Department and many other ancillary services are also available at the hospital.

Although renovated for modern-day patient/physician comfort, much of the townhouse-style flavor remains, including fireplaces and mantels in selected offices. The floor-plan design insures convenience and patient privacy in both the consultation and examination areas. The decor matches the traditional elegance of the surrounding historical homes.

Applications are now being accepted for office space. Interested physicians should call project manager Ray Hayes at (804) 359-1843.

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—A CHARTER MEDICAL CORPORATION FACILITY—

Independent contractor doctors can get tax shelter annuities too.

IRS recently ruled: Compensation earned by an independent contractor doctor—deferred under an annuity-funded agreement with a hospital—isn't includable in his gross income until paid or made available to him *(PLR 8243001, P-H Private Letter Rulings 5286(82); 11-23-82 ITN, page 95).*

Reason: Because the hospital owned the annuity, the doctor had no rights in the annuity. He had nothing more than the hospital's unsecured promise to pay benefits under the deferred compensation agreement.

Result: If you're an independent contractor doctor with a hospital—a radiologist or pathologist, for example—you can agree to defer some of your fees or other current compensation in return for the hospital's agreement to pay income to you or your family at your retirement or death—or termination of your services for other reasons. And the hospital can fund the contract with an insurance-annuity contract.

Typical deferred pay insurance-annuity plan

In lieu of a current compensation contract that pays him $50,000 per year in fees (on top of another $50,000 taxable income from his practice), independent contractor Dr. Holbrook agrees to accept $40,000 in fees each year—and the hospital agrees to pay Dr. Holbrook $25,000 per year in monthly installments for life after his retirement at age 65, provided he stays with the hospital from his present age 45 until age 65.

- If Dr. Holbrook dies before age 65, the hospital will pay Mrs. Holbrook or other beneficiary $25,000 per year for 20 years in monthly installments.
- The hospital will apply for, pay for, and be owner and beneficiary of a $450,000 Whole Life policy on Dr. Holbrook.

Dollars and cents benefits for doctor: Under the deferred pay plan, Dr. Holbrook will get $25,000 before taxes following retirement at age 65—about $16,800 after taxes assuming his other taxable income is $28,000 per year and he files a joint return—$11,300 more than he'd net after taxes if he took the $10,000 now.

- If Dr. Holbrook dies before 65, Mrs. Holbrook (assuming her other taxable income is $20,000 per year) will net her around $16,500 per year—even more if she's head of the household.
- In addition, the contract gives Dr. Holbrook substantial income if, for some reason, he should leave before retirement.

Dollars and cents benefits for hospital: The insurance-annuity-funded deferred compensation plan obligates the hospital to pay Dr. Holbrook about $2,100 per month for life after he reaches age 65.

But the monthly life income from the cash value of the basic contract and the paid up additions purchased by present-scale dividends is about $3,200 per month.

So the hospital benefits to the tune of of $1,100 monthly—or $13,200 per year.

Or, if Dr. Holbrook dies before 65, the hospital will get at least $2,700 per month for 20 years (excluding paid up additions and excess interest) and will pay about $2,100 monthly to Mrs. Holbrook for 20 years.

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"Any drug used in the control of the symptoms of the chronic arthritis must be tolerated for long periods without undue gastric discomfort...From this study it appears that ibuprofen is eminently suitable."[8]

Peptic ulceration and GI bleeding, sometimes severe, have been reported. Rufen® should be given under close supervision to patients with a history of upper GI tract disease.

References: 1. Royer GL, Jr, Moxley TE, Hearron MS, et al: *J Int Med Res* 3:158-171, 1975. 2. Royer, GL, Jr, Moxley TE, Hearron MS, et al: *Curr Therap Res* 17:234-248, 1975. 3. Brackertz B, Busson M: *Brit J Clin Pract* 32:77-80, 1978. 4. Tausch J, Fasching U: *Brit J Clin Pract: A symposium supplement, IXTH European Congress of Rheumatology*, Wiesbaden, Germany, Sept 2-8, 1979, pp 53-61. 5. Lanza FL, Royer GL, Jr, Nelson RS et al: *Dig Dis & Sci* 24:823-828, 1979. 6. Pavelka K, Susta A, Vojtisek A et al: *Rheumatol and Rehab* 12:68-73, 1973. 7. Tretenhahn W: *Brit J Clin Pract: A symposium supplement, IXTH European Congress of Rheumatology*, Wiesbaden, Germany, Sept 2-8, 1979, pp 45-52. 8. Cardoe N: Curr Med Res & Opinion 3:518-520, 1975.

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RUFEN® (ibuprofen) Tablets

INDICATIONS AND USAGE: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in the long-term management of these diseases. Safety and effectiveness have not been established for Functional Class IV rheumatoid arthritis.

Relief of mild to moderate pain. Treatment of primary dysmenorrhea.

CONTRAINDICATIONS: Patients hypersensitive to ibuprofen, or with the syndrome of nasal polyps, angio-edema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory drugs (see WARNINGS).

WARNINGS: Anaphylactoid reactions have occurred in patients hypersensitive to aspirin (see CONTRAINDICATIONS). Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Peptic ulceration, perforation, or gastrointestinal bleeding can end fatally, however, an association has not been established. Rufen should be given under close supervision to patients with a history of upper gastrointestinal tract disease, and only after consulting the ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be attempted. If Rufen must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

PRECAUTIONS: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If developed, discontinue Rufen and administer an ophthalmologic examination.

Fluid retention and edema have been associated with Rufen; caution should be used in patients with a history of cardiac decompensation.

Rufen can inhibit platelet aggregation and prolong bleeding time. Use with caution in patients with intrinsic coagulation defects and those taking anticoagulants.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy, this therapy should be tapered slowly when adding Rufen.

DRUG INTERACTION: *Coumarin-type anticoagulants*: The physician should be cautious when administering Rufen to patients on anticoagulants.

Aspirin: Concomitant use may decrease Rufen blood levels.

PREGNANCY AND NURSING MOTHERS: Rufen should not be taken during pregnancy nor by nursing mothers.

ADVERSE REACTIONS: Incidence greater than 1%. Gastrointestinal: The most frequent adverse reaction is gastrointestinal (4 to 16%). Includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence). Central Nervous System: dizziness*, headache, nervousness. Dermatologic: rash* (including maculopapular type), pruritus. Special Senses: tinnitus. Metabolic: decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS). *Incidence 3% to 9%.

Incidence less than 1 in 100. Gastrointestinal: gastric or duodenal ulcer with bleeding and/or perforation, hemorrhage, melena. Central Nervous System: depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma. Dermatologic: vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome and alopecia. *Special Senses*: hearing loss, amblyopia (blurred and/or diminished vision, scotomata and/or changes in color vision) (see PRECAUTIONS). Hematologic: neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs' positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit. Cardiovascular: congestive heart failure in patients with marginal cardiac function, elevated blood pressure. Allergic: syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasms (see CONTRAINDICATIONS). Renal: acute renal failure in patients with preexisting significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria. Miscellaneous: dry eyes and mouth, gingival ulcers, rhinitis.

Causal relationship unknown. Gastrointestinal: pancreatitis. Central Nervous System: paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri. Dermatologic: toxic epidermal necrolysis, photoallergic skin reactions. Special Senses: conjunctivitis, diplopia, optic neuritis. Hematologic: bleeding episodes. Allergic: serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis. Endocrine: gynecomastia, hypoglycemia. Cardiovascular: arrhythmias (sinus tachycardia, bradycardia, and palpitations). Renal: renal papillary necrosis.

OVERDOSAGE: Acute overdosage: the stomach should be emptied. Rufen is acidic and excreted in the urine, alkaline diuresis may benefit.

DOSAGE AND ADMINISTRATION: Rheumatoid arthritis and osteoarthritis, including flareups of chronic disease: Suggested dosage 400 mg t.i.d. or q.i.d.

Dysmenorrhea: 400 mg every 4 hours as necessary.

Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for the relief of pain. *Do not exceed 2,400 mg per day.*

CAUTION: Federal law prohibits dispensing without prescription.

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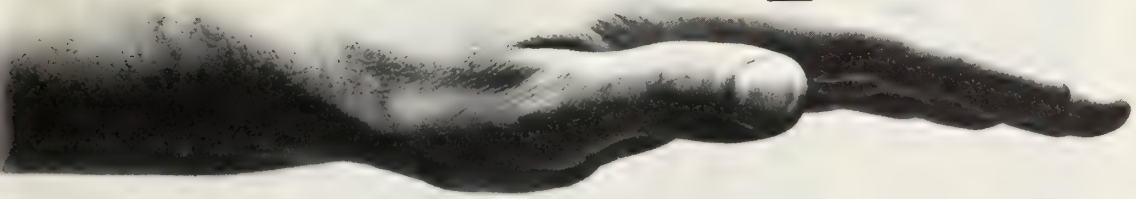

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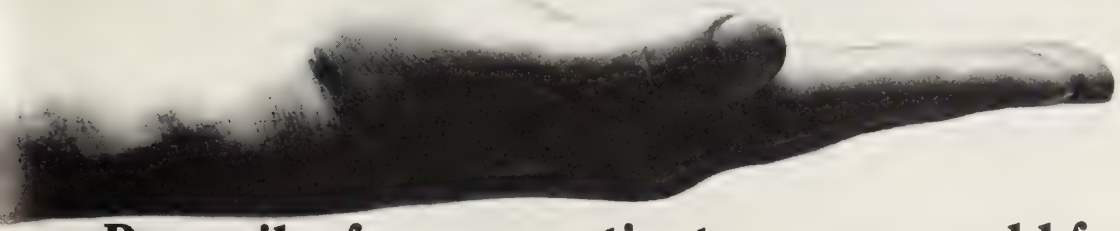

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It is this focus on recognition and acceptance that works to end the repetition of relapse and allows the family to continue toward total alcoholism recovery.

But the Relapse Program is just one of many family-oriented programs designed by Peninsula Psychiatric Hospital to treat all forms of chemical dependency. Here is a brief profile of these exceptional support programs:

The Adult Chemical Dependency Treatment Program deals with individual patient needs in a non-psychiatric approach that involves intense hospital care and patient/family education and counseling. Emphasis is on communication and non-chemical coping skills without the use of psychotropic drugs.

The Adolescent Chemical Dependency Treatment Program places special emphasis on the adolescent and the family, with therapy designed to help restructure the family unit and aid in the recovery of all. The full services of Peninsula Academy allow for continued education while the patient undergoes treatment.

The family program for Adolescent and Adult Chemical Dependency focuses on helping concerned individuals adjust to living with another's alcohol or drug problem. The program's non-psychiatric approach emphasizes family education to the problem, enhanced by individual and group therapy with an aim toward positive development of the whole self.

As with all Peninsula Psychiatric Hospital's programs, there are no eligibility restrictions with regard to race, religion, sex, residency or disability. For details on any of our Chemical Dependency Treatment Programs, telephone **Hampton (804) 722-2504, Norfolk (804) 622-5577, Richmond (804) 796-2016.**

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An added complication... in the treatment of bacterial bronchitis*

Increasing incidence of ampicillin resistance in Haemophilus influenzae
Ampicillin Resistant Haemophilus influenzae
H. influenzae
S. pneumoniae

Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor
Pulvules®, 250 and 500 mg

Brief Summary. Consult the package literature for prescribing information

Indications and Usage. Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings. IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out

Precautions: *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor® (cefaclor, Lilly) is administered to a nursing woman

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established

Adverse Reactions. Adverse effects considered related to therapy with Ceclor are uncommon and are listed below

Gastrointestinal symptoms occur in about 2-5 percent of patients and include diarrhea (1 in 70)

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely

Hypersensitivity reactions have been reported in about 1-5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients)

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40)

Hematopoietic—Transient fluctuations in leukocyte count predominantly lymphocytosis occurring in infants and young children (1 in 40)

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200)

Jb17-82R

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[1]

Note. Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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7. Data on file, Eli Lilly and Company.
8. Principles and Practice of Infectious Diseases (edited by G. L. Mandell, R. G. Douglas, Jr., and J. E. Bennett), p. 487. New York: John Wiley & Sons, 1979

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BOOKS

FIG. 16-5
Lateral ankle sprains. A "single-ligament sprain" occurs when the player's plantar-flexed ankle is inverted, tearing the anterior talofibular ligament. If the inversion continues when the whole foot has reached the ground, the calcaneal-fibular ligament rips, producing a "double-ligament sprain."

The Injured Athlete, by Daniel N. Kulund, MD. Illustrated by Ronald J. Ervin. Philadelphia, J. B. Lippincott Company, 1982. 544 pages, 223 illustrations, nine tables. Hardcover, $42.50.

Dr. Kulund, who is assistant professor of orthopedic surgery, health and physical education at the University of Virginia, has chosen a subject that will have increasing relevance as the fitness boom continues to expand.

His book is extensive in coverage, with 16 chapters. There is pertinent discussion of the role of the athletic physician, handicapped athletes, drugs, non-musculoskeletal medical problems, training principles, fitness evaluation, cardiac considerations, warm-up, athletic trainer, taping, psychological factors, head, face and neck injuries, as well as the standard orthopedic problems.

The early chapters on the athletic physician, fitness evaluation, cardiac considerations and athletic training are particularly helpful. In the orthopedic sections the informational quality of the knee and hand areas is exceptional.

This is, in sum, a book of much merit. It will be helpful to any physician who treats injured athletes.

ROBERT P. NIRSCHL, MD
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Illustration at left from *The Injured Athlete*. © J. B. Lippincott. Reproduced by permission.

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The dosage form children like

Antiminth is available as a pleasant tasting, caramel-flavored oral suspension. Effective in just

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Warnings

Usage in Pregnancy Reproduction studies have been performed in animals and there was no evidence of propensity for harm to the fetus. The relevance to the human is not known.

There is no experience in pregnant women who have received this drug.

The drug has not been extensively studied in children under two years; therefore, in the treatment of children under the age of two years, the relative benefit/risk should be considered.

Precautions

Minor transient elevations of SGOT have occurred in a small percentage of patients. Therefore, this drug should be used with caution in patients with pre-existing liver dysfunction.

Adverse Reactions

The most frequently encountered adverse reactions are related to the gastrointestinal system.

Gastrointestinal and hepatic reactions: anorexia, nausea, vomiting, gastralgia, abdominal cramps, diarrhea and tenesmus, transient elevation of SGOT.

CNS reactions: headache, dizziness, drowsiness, and insomnia. Skin reactions: rashes.

Dosage and Administration

Children and Adults Antiminth Oral Suspension (50 mg of pyrantel base/ml) should be administered in a single dose of 11 mg of pyrantel base per kg of body weight (or 5 mg/lb.); maximum total dose 1 gram. This corresponds to a simplified dosage regimen of 1 ml of Antiminth per 10 lb. of body weight. (One teaspoonful = 5 ml.)

Antiminth (pyrantel pamoate) Oral Suspension may be administered without regard to ingestion of food or time of day, and purging is not necessary prior to, during, or after therapy. It may be taken with milk or fruit juices.

References 1. Pitts NE, Migliardi JR: *Clinical Pediatrics* 13:87, 1974. **2.** Modell W: *Drugs of Choice* 1980-1981. C. V. Mosby Co., St. Louis, 1980, p. 362. **3.** Goodman LS, Gilman A: *The Pharmacologic Basis of Therapeutics*, 6th edition, MacMillan Publishing Co., Inc., New York, 1980, p. 1032.

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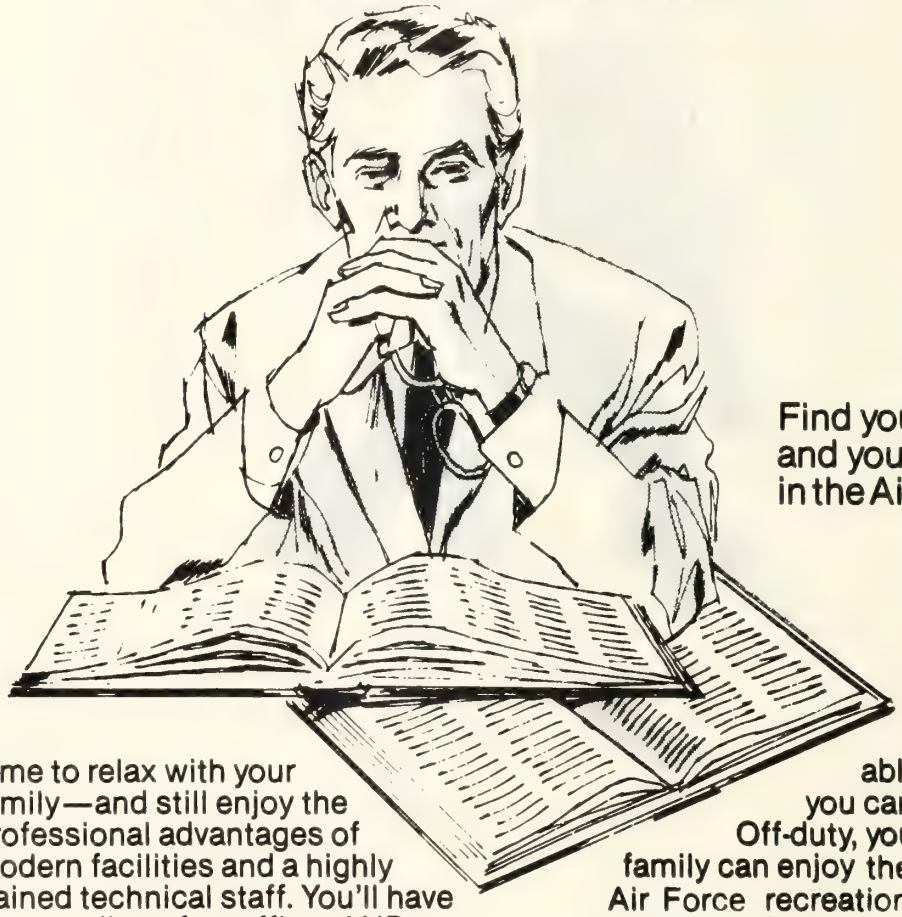

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Tumors Compressing the Optic Nerve: Diagnosis and Surgical Results

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Study of a small series shows that modern neurosurgical techniques offer a minimum of risk in resection of intracranial meningiomas involving the anterior visual system, with improvement of vision possible despite long compression of the optic nerve. Cautious attention by the physician to a patient's progressive visual loss without a clear ocular explanation could further reduce residual visual loss from these tumors.

MENINGIOMAS arising from the tuberculum sellae, planum sphenoidale and medial sphenoid wing often compress the optic nerve and chiasm. Insidious, progressive visual loss parallels the slow growth rate of these intracranial tumors. Additional neurologic signs are infrequent. Detection is often delayed, and the tumors may attain a size surprising in the absence of neurological signs. These meningiomas are precariously situated at the base of the brain so delay in diagnosis places in jeopardy to neoplastic encroachment the hypothalamus, carotid arteries and the remaining, functioning optic nerve.

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Submitted 3-15-82.

After recognition, a formidable question for the clinician is the risk to life and to adjacent brain and visual system posed by removal of these tumors. How do these complications compare to eventual blindness and neurological impairment from growth of the tumor? Are there any other benefits, especially for reversal of impaired vision? With modern advances in diagnostic and neurosurgical techniques, we sought to answer these questions by reviewing our experience in recent years with intracranial meningiomas compressing the anterior visual system.

Case Reports

Over the six-year period 1975-1980, 12 patients (Table 1) were encountered with meningiomas arising from the tuberculum sellae (#1-9), anterior clinoid (#10) and medial sphenoid wing (#11,12).

Visual symptoms were the presenting feature in each patient. Duration of visual loss ranged from three months to 15 years prior to diagnosis, occurring for one year or longer in nine patients. Because the loss of vision was painless and usually confined to one eye, symptoms of impaired vision were often subtle. For example, three patients complained of distorted letters while reading. One patient first noticed his altered vision when he was unable to see the crosshairs of a gunsight. Another patient's first clue to monocular dysfunction was difficulty viewing the bottom of a wine bottle while peering down its neck. One patient, a greenskeeper, noted that the grass appeared more blue than green with one eye compared to the other eye. A truck driver was brought by his wife after receiving his second traffic citation for driving too slowly. The subtle, progressive visual loss was often not voiced by the patient or appreciated by the general physician. In some instances the visual deficit was falsely attributed to a long-standing, stable, optic neuropathy or retinopathy even though some progression had ensued.

By the time of our examination severe loss of visual acuity was present in nearly every patient. All patients had dyschromatopsia demonstrable by color plate testing, and many had an afferent pupillary defect with the swinging flashlight test for optic nerve dysfunction. Definite optic pallor was observed in eight of 16 affected eyes. Visual field defects included a bitemporal depression in five patients, monocular suppression in five patients, a binasal defect in one patient and an incongruous homonymous defect in another patient. Limited gaze of the ocular muscles and mild proptosis of one eye occurred only in the two patients with sphenoid wing meningiomas; nonetheless, they did not complain of diplopia or proptosis, and these ocular signs were so slight that they were not noted by physicians examining the patients for other reasons. None of the patients had additional signs or symptoms of nervous system involvement.

Computerized tomographic (CT) scans of the head, some requiring enhancement, were typical for a meningioma in each patient. These features often included a round mass with uniform density and homogeneous enhancement following addition of contrast (Figure 1). Such lesions were adjacent to the bony structures near the intracranial segment of the optic nerve. In one patient the unenhanced scan did not show any clear abnormal density until contrast was used (Figure 2), and we recommend that all patients having a CT scan for undiagnosed loss of vision in one eye undergo a contrast-enhanced CT scan. Calcifications were seen in some tumors (Figure 3). Roentgenogram of the skull showed hyperostosis of bone in only one patient, a feature supposedly typical of basal meningiomas but infrequent in our patients. To confirm the clinical and CT scan impression and to delineate major arterial feeders, all patients underwent cerebral angiography. There were no complications due to angiography, and usually the expected homogeneous hypervascularity of a meningioma was found.

Surgical Results

All patients underwent craniotomy. No deaths occurred. Temporary diabetes insipidus developed in one patient and anosmia in another patient. A seizure was reported five years after surgery in another patient. Tumor removal was incomplete in two patients. Because of the partial resection, irradiation was delivered. No recurrence has been noted to date, but one patient lost an already blind eye due to complications of the irradiation.

Table 1. Results of Craniotimy for Meningioma of the Optic Nerve in 12 Patients from 1975 to 1980

			Preoperative				Postoperative		
			VA		Optic pallor		VA		
#	Age/Sex	Duration Symptoms	OD	OS	OD	OS	OD	OS	CT Diagnostic
1	64 F	15 years	20/40	NLP	–	+	20/20	NLP	+
2	63 M	6 months	20/800	HM			20/20	20/25	+
3	59 M	1 year	20/40	HM	–		20/20	20/200	+
4	51 M	15 months	*	20/400	–	tr	*	20/20	+
5	31 F	3 years	NLP	20/70	+	tr	NLP	20/20	+
6	71 F	15 years	*	20/70	–	tr	*	20/400	+
7	23 F	3–4 months	*	NLP	–	+	*	NLP	+
8	43 F	14 years	20/200	*	+	–	20/20	*	+
9	29 F	9 months	CF	*	+	tr	20/50	*	+
10	54 F	6 years	HM	*	+	–	NLP	*	+
11	50 M	2 years	NLP	*	+	–	NLP	*	+
12	40 F	1 year	HM	*	+	–	20/400	*	+

CF = finger counting; HM = hand motion; NLP = no light perception; Tr = trace; + = positive; – = negative or absent; * = visual acuity 20/30 or better pre- and post-operatively.

Postoperatively, visual acuity improved in nine of 16 affected eyes (56%), remained the same in six of 16 (37%) and was worse in one patient (Table 1, #6). Two of four patients with bilateral visual loss had improvement of visual acuity in both eyes. A better visual acuity was obtained in two of eight eyes with definite optic pallor and two of four eyes with a trace of paleness of the optic discs. Visual field deficits distinctly improved in four patients.

Comments

Cushing and Eisenhardt were the first to distinguish clinically between intrasellar and suprasellar tumors.[1] The triad of optic atrophy, bitemporal visual field defects and a normal sella turcica without a clinical endocrinopathy subsequently became known as Cushing's syndrome of the chiasm.[2] By far the most common cause of this syndrome is a meningioma, but in adults a differential diagnosis of Cushing's syndrome of the chiasm includes giant aneurysms, craniopharyngiomas and several rarer entities. In the six-year interval reported here, we also encountered three giant aneurysms and two craniopharyngiomas. A clinical distinction between these masses is not possible but is often attainable by radiological study, usually CT scan alone. An arteriogram is necessary to definitely exclude an aneurysm. The lack of complications and diagnostic accuracy of the neuroradiologic procedures in these 12 patients speaks for the safety and value of these modern techniques.

Today, with CT scanning, the diagnosis of a suprasellar tumor is possible early in the period of decreased visual acuity rather than with the late development of optic atrophy. Not infrequently the difficulty in early diagnosis involves the patient's or physician's lack of appreciation of the potential seriousness of slowly progressive visual loss.[3,4] In past series, misdiagnosis of the early visual loss was not uncommon and included diagnostic categories such as optic neuritis, low pressure glaucoma and maculopathy.[5-9] Severe loss of vision in one eye or involvement of the second eye usually brought the patient to our diagnostic attention. In only three patients did concern for an appropriate diagnosis initiate a diagnostic investigation within one year.

In 1972 Knight, Hoyt and Wilson reported their experience with four techniques to evaluate optic nerve function clinically: visual acuity, color plate recognition, visual fields and pupillary testing with the swinging flashlight test.[10] In the absence of recognizable disease of the fundus, incipient optic nerve dysfunction can be suspected from abnormalities in any, and often all, of these tests. All of the 16 symptomatic eyes of our 12 patients had abnormalities in each test of this battery. It is important to realize that pallor of the optic disc is a late sign of optic nerve dysfunction due to intracranial disease.[3]

Fig. 1. Homogeneous appearance of tuberculum sellae meningioma on CT scan with contrast enhancement.

Fig. 2. Sphenoid wing meningioma detected with contrast enhancement after scan without contrast did not define it.

In fact, the optic disc head appeared normal in four eyes, was only slightly pale in four more eyes and definitely pale in just half (eight) of the patients in this series. Duration of symptoms in patients with definite pallor extended between nine months and 15 years. The late appearance of optic pallor supports the notion that compression of the optic nerve and dysfunction due to physiologic conduction block of optic nerve impulses precedes by months the irreversible destruction of optic nerve axons that leads to optic atrophy.[11]

A common operative approach to these intracranial tumors in the past was a right frontal craniotomy.[12] Rather than arbitrarily operate on the right, we perform a craniotomy on the side with the most severe visual loss, as this represents the site of greatest tumor bulk. Other surgeons approach the side with the best vision in order to fully view and protect the less affected optic nerve, but this approach seems to increase the possibility of incomplete removal. With large, midline tumors, a bifrontal craniotomy is sometimes performed. Generally, the tumor is easily reached by a lateral subfrontal exposure just in front of the sphenoid wing, which can be traced medially to the intracranial optic nerve. A unilateral craniotomy is advised whenever possible to preserve at least one olfactory nerve and avoid the sometimes troublesome loss of smell and occasionally taste that follows bilateral sacrifice of the olfactory nerve. In recent years blood supply to the often vascular meningioma was interrupted with a surgical laser, which is quite effective in maintaining a bloodless field. Internal decompression of the mass is done with the aid of bipolar coagulation and small ring currettes. The tumor is carefully removed in painstaking piecemeal fashion even if this requires many hours of general anesthesia. Microdissectors and magnification techniques allow the edges of the meningioma to be dissected from the optic nerve, chiasm, pituitary stalk and blood vessels. Involved dura mater, especially over the tuberculum sella, and hyperostotic bone is removed with a diamond burr.

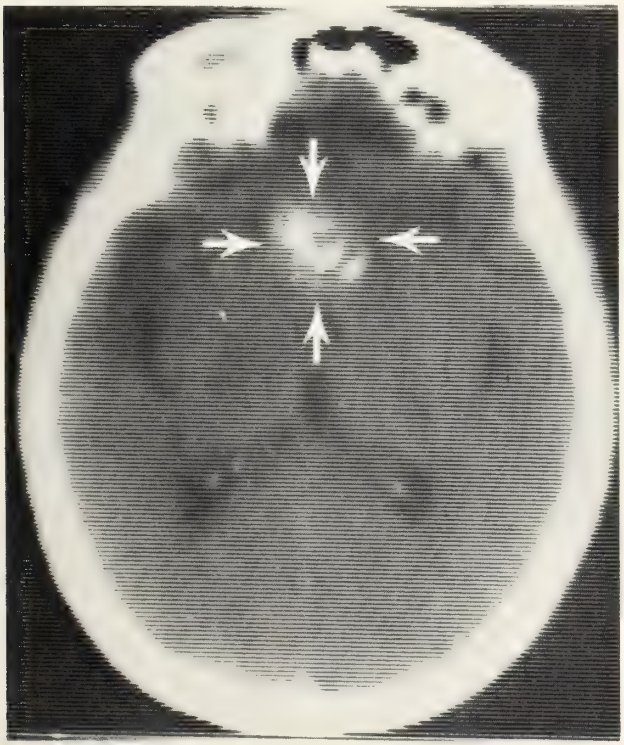

Fig. 3. Calcification noted on CT scan in a tuberculum sellae meningioma prior to contrast enhancement.

Mortality rates in series spanning 1940 to 1972,[7] 1940 to 1978,[8] and others[9,13,14] were 7% to 11%. In one series covering 1960 to 1978, one delayed death occurred in a group of 19 patients.[15] The principal complications were arterial or cerebral damage occurring during surgery. An 8% recurrence rate was noted in one series that involved 83 meningiomas[7] and 8.8% in a series of ten patients.[14] Morbidity included diabetes insipidus (19% to 23%), infections, CSF leak, hematoma, cerebral edema and seizures.[7,8] In several reports visual acuity improved in 60% of cases following surgery,[7-9] 28% in another series.[15]

In our short series there was no mortality and minimal morbidity. In only two patients was removal incomplete. These two patients received postoperative irradiation in accordance with a report by Wara and associates.[16,17] No recurrence has been reported to date, but one patient lost an already blind eye due to complications of irradiation. Visual acuity improved or remained stable in 15 of 16 eyes. Complete removal of one tumor symptomatic over 15 years resulted in additional impairment of the involved optic nerve. Those eyes which exhibited no or very little optic disc pallor were most likely to show recovery in visual acuity postoperatively, especially if diagnosed within one year. The recovery was usually detectable shortly after surgery. Hence, decompression reversed an apparent conduction block and permitted a rapid return in visual function.

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COMMENT

Drs. Waybright, Selhorst, Young and Harbison are correct: The diagnosis of progressive visual problems can be very difficult, impeding the ophthalmologist's early referral of patients. Awareness that a tentative ocular explanation may be in error, serial examinations, visual fields, color-vision testing, and careful observation of the pupils for the Marcus Gunn phenomenon via the swinging flashlight test are useful clinical tools for suspecting life-threatening disease. However, the definitive diagnosis is made by CT scans of the head with contrast, followed by arteriography to rule out vascular lesions.

How many normal CT scans equal one positive scan for this subtle, progressive tumor which surgery can give such an excellent prognosis for life and vision? A positive CT scan on one of my patients is worth a googol of normal CT scans, when I have used thought and care before requesting each scan. While cost effectiveness is chic, it has more value in a factory than in a physician's office. Certainly I shall be requesting more CT scans now that I have read this article.

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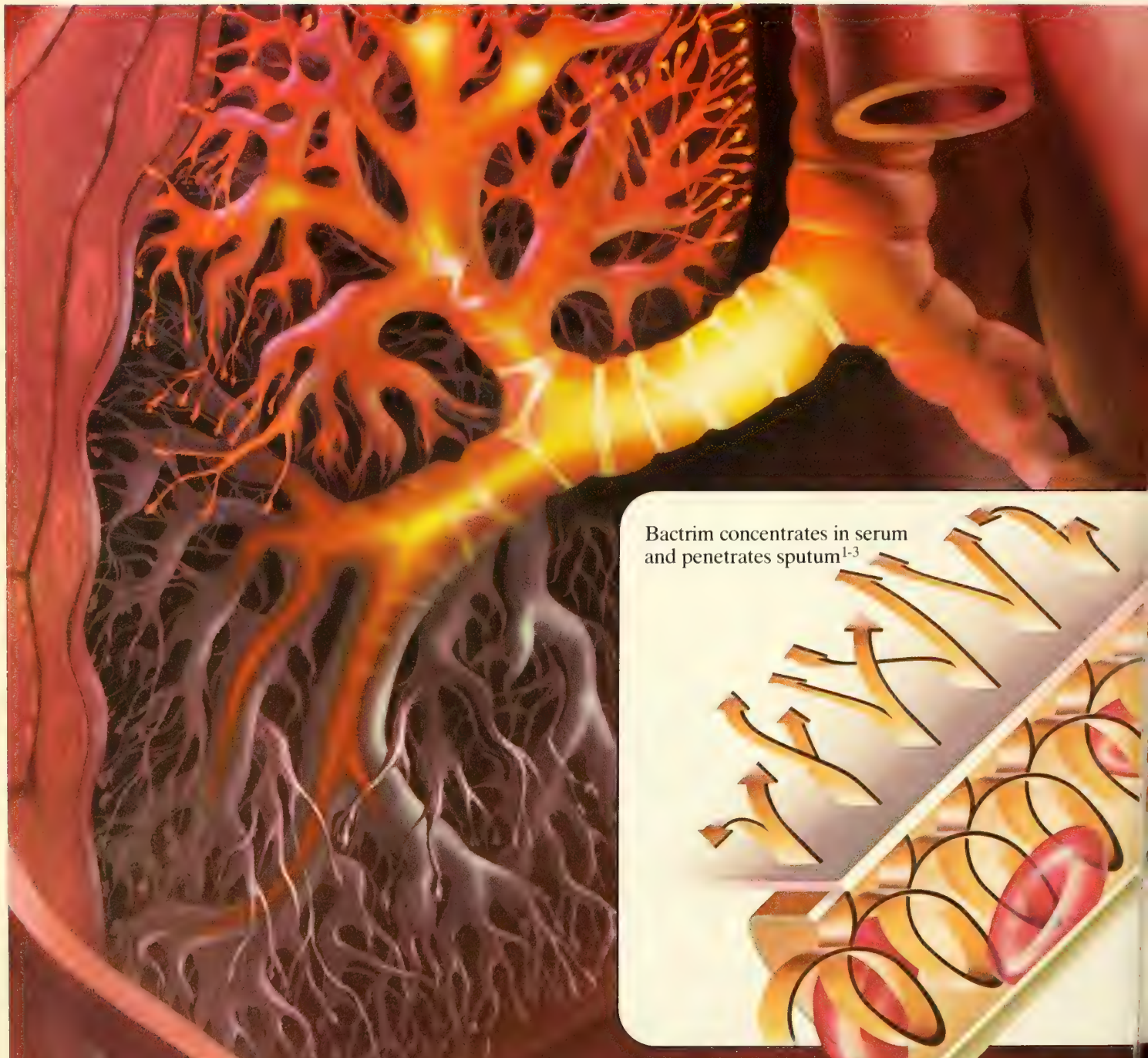

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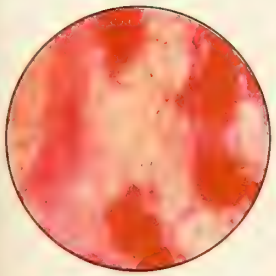
major pathogens of chronic bronchitis*

Bactrim clears sputum of susceptible bacteria

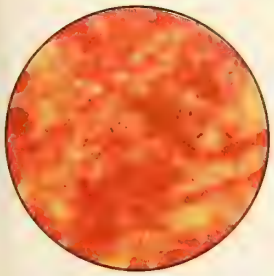
In sputum cultures from patients with acute exacerbations of chronic bronchitis, *H. influenzae* and *S. pneumoniae* are isolated more often than any other pathogens.[4,5] One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens.[5]

Bactrim is effective *in vitro* against most strains of both *S. pneumoniae* and *H. influenzae*—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.[6]

attacks *H. influenzae*—even ampicillin-resistant strains



attacks *S. pneumoniae*



Bactrim reduces coughing and sputum production

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Bactrim also proved more effective than tetracyclines in 10 clinical trials involving nearly 700 patients.[10] Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

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*Due to susceptible organisms. **Please see next page for summary of product information.**

Bactrim™

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Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy. Teratogenic Effects. Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

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Grand Rounds: North American Blastomycosis

From the Department of Medicine
University of Virginia School of Medicine
Discussed by W. Michael Scheld, MD

Presentation of Case

Dr. Robert Wells: A 56-year-old, black, male, water-well driller from Fairfield, Virginia, was in excellent health until February 1981 when he noted the onset of a dry cough, chest tightness, fever and chills. His cough became progressively more productive of white sputum over the ensuing weeks, and he experienced a weight loss of 10-12 lbs. Because a chest X-ray showed a left upper lobe infiltrate, he was treated by his local physician for pneumonia with a two-week course of oral penicillin followed by a two-week course of a sulfa antibiotic. Despite this treatment, he did not improve; his sputum became blood-streaked, and his voice became hoarse. He was seen in late April 1981 by an otolaryngologist, who detected a lesion on the right true vocal cord.

The patient was referred to the University of Virginia Medical Center on 10 May 1981. Chest X-ray on admission revealed a lingular infiltrate; tomography showed compression of the lingular bronchus by a mass (Figs. 1A, 1B, 1C). Bronchoscopy showed 50% concentric narrowing of the lingular bronchus, but washings, brushings and biopsy for culture and pathology were negative. Liver-spleen scan and brain scan were normal. Bone scan showed increased uptake at vertebral body T_{11}, and thoracic spine X-rays demonstrated degenerative changes in the region suggestive of old trauma. A presumptive diagnosis of carcinoma of the lung was made, and the patient was scheduled for a second bronchoscopy on 26 May 1981.

On physical examination when he was readmitted to the hospital in late May 1981, the patient was described as a thin, middle-aged man in no distress. His blood pressure was 155/95 mm Hg, pulse 80/min and regular, respirations 16/min; he was afebrile. A raised, verrucous lesion, 1.0 × 1.5 cm, was seen on the right cheek; the lesion had appeared during the preceding week. The head and neck examination was otherwise notable only for a hoarse voice. The lungs were clear to auscultation and percussion, and the heart sounds were normal except for an S_4 gallop. There was no hepatosplenomegaly. The prostate was moderately enlarged but not tender. There was no localized tenderness on percussion of the spine. The remainder of the physical examination was normal.

Edited by Richard J. Gualtieri, MD, and Marcia Day Finney. Address correspondence to Mrs. Finney at Box 466, Department of Medicine, University of Virginia, Charlottesville VA 22908.

Presented 6-11-81.

Laboratory work on admission revealed a hematocrit of 42.8%, WBC 8400/mm^3 (1% bands, 60% polymorphonuclear leukocytes, 32% lymphocytes, 6% monocytes, 1% eosinophils), erythrocyte sedimentation rate 5 mm/hr. All other laboratory values, including a platelet count, metabolic profile, serum enzyme and urinalysis, were within normal limits. A repeat chest X-ray showed a patchy infiltrate in the lingula and upper left lobe.

The second bronchoscopic examination yielded no new information. However, biopsy of the lesion on the patient's right cheek demonstrated, on methenamine silver staining, broad-based, wide-pore budding yeast typical of *Blastomyces dermatitidis*. A diagnosis was made of disseminated North American blastomycosis, with involvement of the lung, skin, and, possibly, larynx.

The patient was advised of the standard therapy with amphotericin B and of an optional therapy with the new antifungal agent, ketoconazole; he chose to begin treatment with ketoconazole, 800 mg orally per day, and was enrolled in an ongoing clinical study of the efficacy of this agent in disseminated blastomycosis. The patient initially experienced some nausea as a result of the medication, but he has otherwise tolerated it well. He was discharged from the hospital after one week of observation. Shortly after his discharge, cultures of the bronchial washings, skin biopsy, and postprostatic-massage urine grew *Blastomyces dermatitidis*.

Dr. Edward W. Hook: I am curious why you cultured the urine for *Blastomyces* when the patient had a normal urinary sediment and a prostate that was only slightly enlarged and not tender.

Dr. Wells: We obtained the urine specimen after we knew that the silver stains of the skin lesion showed yeasts. We suspected blastomycosis and considered the possibility of prostatic involvement. Therefore, the prostate was massaged and a urine specimen collected and sent for culture.

Discussion

Dr. W. Michael Scheld: This patient's presentation of disseminated blastomycosis is typical. Typical, too, was the fact that he was ill for three to four months before a diagnosis was made. When the

Figs. 1A, 1B, 1C: Admission chest radiographs. At top, AP view and, at center, lateral view, both showing lingular infiltrate. Bottom, tomogram demonstrating left main-stem bronchus (blunt arrow), left upper-lobe bronchus (large arrow), and concentric narrowing of lingular bronchus (small arrow).

diagnosis was finally made, we were able to document involvement of the skin, lung, and genitourinary tract; his larynx is also probably involved. There is insufficient evidence to suggest bone involvement. Had the skin lesion not appeared, I suspect that we would have made the diagnosis of a curable fungus infection at pneumonectomy; as Dr. Wells mentioned in his presentation, compression of the lingular bronchus raised the prospect of a neoplasm.

The patient's laboratory values are not, however, typical of chronic blastomycosis, for he was not anemic and had a normal sedimentation rate.

Of the three major mycoses endemic in the United States, North American blastomycosis is the only one initially described in this country. In 1894, T. C. Gilchrist, a pathologist at Johns Hopkins University, was asked by a Philadelphia physician to review a skin biopsy. Gilchrist studied the specimen and noted "peculiar corpuscles", which he at first thought were protozoa but later correctly identified as budding yeasts. He presented his findings in May 1894 at the annual meeting of the American Dermatology Association. This was the first demonstration of a yeast infection pathogenic for man. Four years later, Gilchrist and Stokes reported a second case; this time they were able to isolate the organism from the patient's skin lesion. They named the organism *Blastomyces dermatitidis* and were able to infect dogs with it.[1] It was thought that the disease was limited pathogenically to the skin.

Between 1900 and 1920 a number of cases of blastomycosis were reported from Chicago, and the illness became known colloquially as "Chicago disease." In 1916, a single issue of the *Archives of Internal Medicine* contained a report of a series of 36 cases from Chicago's Cook County Hospital and reports of another 11 cases.[2] By this time the disease was no longer regarded as affecting only the skin, for all of these reports described the systemic form of disseminated blastomycosis with multiple organ involvement. The Chicago cases had a mortality rate of 92%. In the report of the large series of cases from Chicago, the authors noted that the fungus was isolated from wood in rotting dwellings and tenements in South Chicago. They also described a diagnostically useful potassium hydroxide preparation.

In 1951, Schwarz and Baum delineated the pathogenesis of blastomycosis and demonstrated that virtually all cases are due to a primary pulmonary focus.[3] When stilbamidine was introduced in 1953, we had for the first time a cure for disseminated blastomycosis. In 1956, our armamentarium against the disease was strengthened by the introduction of amphotericin B.

With Emmons's description of numerous cases of blastomycosis in Africa came the recognition that this disease occurs worldwide; the name "North American blastomycosis" is therefore imprecise and misleading. In 1967, McDonough and Lewis described the perfect state or sexual stage of the *Blastomyces* fungus. The most recent advances have come in our ability to treat blastomycosis. We are now evaluating ketoconazole, a new antifungal agent which may alter dramatically the outlook for many patients with disseminated blastomycosis.

The epidemiology of blastomycosis remains poorly understood because we lack a specific or sensitive skin test.[4] The incidence of the disease is not known but is presumed to be less than that of histoplasmosis. The disease most often affects persons in middle age; 87% of patients are between 20 and 69 years of age, with about 40% of patients over age 50. Males are affected more often than females, with the ratio as high as 9 : 1 in some series. The disease occurs predominantly in North America, but the lack of a skin test for identification of previously infected individuals precludes our being able to map out the disease's hyperendemic area as we have been able to do for both histoplasmosis and coccidioidomycosis.

We know little about the natural habitat of *Blastomyces*, for the organism is virtually impossible to isolate from the soil. In one study carried out in Augusta, Georgia, in 1963, only 10 of 400 soil samples were positive for *Blastomyces*, with organisms found in such diverse locations as chicken houses, mule stalls, abandoned kitchens, and rabbit pens.[5] Interestingly, the organism will grow luxuriously when inoculated into sterile soil in the laboratory, but when the hyphae are introduced into raw soil in the lab, they promptly lyse. This contrasts sharply with *Histoplasma*, which grows readily in raw soil. Only once has *Blastomyces* been isolated from bird guano,[6] whereas *Histoplasma* and *Cryptococcus* grow very well in this medium.

We presume that *Blastomyces* enters man via the lung. The majority of cases of blastomycosis are due to inhalation of conidia, although direct primary skin inoculation occurs occasionally as the result of laboratory accidents (or, in one case, it followed the bite of an infected pet dog). There has been one documented case of venereal transmission through infected prostatic secretions; the partner developed endometritis with a tubo-ovarian abscess.[4] No occupation seems to predispose persons to infection, although many patients report having contact with

soil. In the north central United States, blastomycosis occurs commonly in hunters, particularly those in contact with bird dogs. The dogs develop the same illness as man except that they experience more ocular involvement. We suspect that the hunters and their dogs come into contact with the fungus in wooded areas. There seems to be a fairly long incubation period, for, typically, hunters develop clinical blastomycosis in January or February, 30-45 days after the close of the bird hunting season in Wisconsin and Minnesota.[7]

Four "epidemics" of blastomycosis are on record,[8] all probably common-source outbreaks since man-to-man transmission of the disease has never been demontrated.

Like *Histoplasma capsulatum* and *Paracoccidioides brasiliensis*, *B dermatitidis* is a thermal dimorphic fungus. Thermal dimorphic organisms grow as mycelia in nature or at room temperature (25°C) but grow as yeasts in mammalian hosts or at 37°C in the laboratory. The slender conidiophores, approximately one μ in size, are demonstrable microscopically when the organism is teased out of the culture. The conidiophores bear dumbbell-shaped conidia 2-10 μ in size (Fig. 2); these are the particles which infect man. Although the mycelium of *B dermatitidis* may be distinguished from that of *H capsulatum* only with difficulty, it is easy to distinguish these two fungi in their yeast phases. The mother yeast of *Blastomyces* is quite large (8-15 μ), with a single bud through a wide-based pore about 4-5 μ wide, compared to those of *Candida* and *Histoplasma*. *Blastomyces* also has a doubly contoured refractile cell wall, which enables one to differentiate it from *Cryptococcus* in histologic preparations. It is important to note that *Blastomyces* is the only multinucleate yeast pathogenic for man; *Cryptococcus*, *Histoplasma* and *Candida* are all uninucleate. Finally, *P brasiliensis*, the etiologic agent of South American blastomycosis, differs from *Blastomyces* in that the former has small budding yeast forms in a characteristic spoke-wheel arrangement.

In terms of pathology, there are several features characteristic of infection with *Blastomyces*. The typical lesion is a mixed granulomatous and suppurative inflammation.[9] Suppuration is only rarely seen in other fungal illnesses. The granulomas are epithelioid granulomas with giant cells, and they very seldom caseate; these lesions may be confused with those of sarcoidosis. The characteristic skin lesions show pseudoepitheliomatous hyperplasia, granulomas and microabscesses in the dermis and epidermis, with yeast forms present. Figure 3 is a

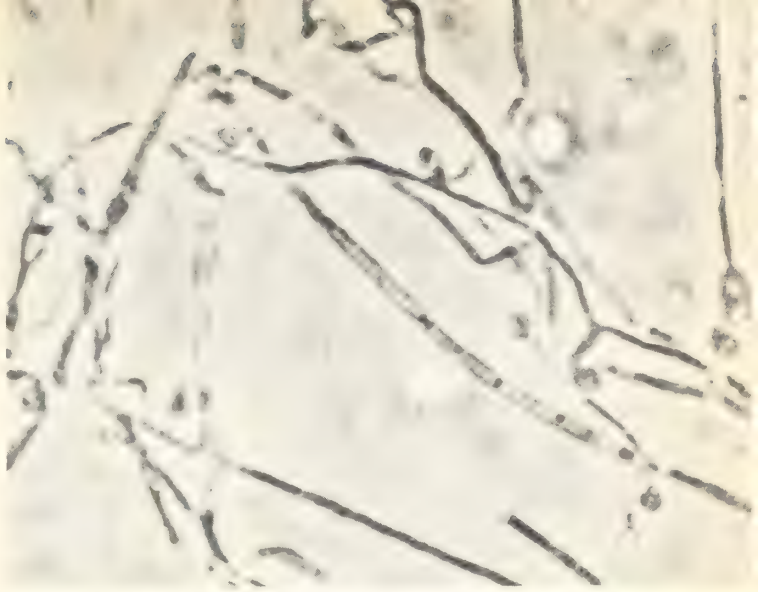


Fig. 2. Photomicrograph of *Blastomyces dermatitidis* in culture at 25°C, mycelial phase. Note slender conidiophores bearing dumbbell-shaped conidia.

Fig. 3. Photomicrograph from biopsy of blastomycotic skin lesion (H&E stain). Note pseudoepitheliomatous hyperplasia and granuloma formation.

histologic section of the skin biopsy from the patient discussed today; it is essentially diagnostic for North American blastomycosis. Virtually the same picture is seen in the lungs with a variable number of yeasts contained in the lesions. One-third of patients with blastomycosis have ulcerative bronchitis; 20% of the pulmonary infiltrates cavitate, and pleural fibrosis is very commonly seen at autopsy. Rarely, the central nervous system is involved, in which case yeasts with polymorphonuclear leukocytes are found in the subarachnoid space; it is unusual to see granulomas.

Dr. George Sarosi and colleagues at the Universi-

ty of Minnesota have investigated the pathogenesis of blastomycosis.[4,9-11] Infection occurs when the *Blastomyces* spores are inhaled. Some patients develope acute pneumonia, some display no symptoms whatsoever. Many patients will recover from the pneumonia and remain well, but progressive pulmonary infection occurs in some and others may experience initial recovery followed by an endogenous reactivation of disease with chronic pulmonary or extrapulmonary involvement. The infection may disseminate to the skin or to any other organ of the body. The parallel with tuberculosis is unmistakable. What determines the outcome of the disease in untreated patients is wholly unknown.

Blastomycosis is protean in its clinical manifestations, ranging from an asymptomatic pulmonary infection to a rapidly fatal systemic illness. The acute pneumonic form resembles community-acquired pneumonias due to other more common pathogens. Patients develop a high fever (sometimes up to 105°F) with rigors, sweats, a cough productive of purulent sputum, and significant myalgias and arthralgias; the illness usually lasts less than three weeks. The physical examination may be entirely normal or may reveal signs of pulmonary consolidation. Roentgenographic findings vary greatly and may or may not include pulmonary consolidation. Occasionally, a miliary pattern is observed, and the illness is confused with miliary tuberculosis. In some cases, the clinical picture resembles that of acute respiratory distress syndrome, although pleural effusions are highly unusual. With this presentation, the differential diagnosis is broad, including bacterial, viral and other fungal infections.[4,10,12-14] For some patients, the illness will prove fatal, while in others the lesions will clear only to reappear at a later date. The outcome cannot be predicted on the basis of laboratory or clinical parameters.

The more common form of blastomycosis presents as a progressive pulmonary disease with or without metastases and represents a reactivation of an initial focus of infection.[4,12,15] The illness resembles tuberculosis or chronic histoplasmosis, with insidious onset of low-grade fever, chills and sweats. Patients also experience weight loss, anorexia and myalgias, and they develop a cough productive of purulent sputum. Gross hemoptysis is rare, but blood-streaked sputum is commonly seen. The patient discussed today had this form of blastomycosis.

Lymphohematogenous dissemination of infection most often involves the skin, bone and male genitourinary tract.[4] Skin involvement is most common, affecting 20-40% of patients on presentation; 80% of patients will eventually demonstrate skin involvement. Usually, the patient develops a slowly expanding skin lesion which is neither painful nor pruritic and which is located on an exposed area of the body such as the face, hand, arm or scalp. Beginning as a subcutaneous nodule, the lesion ulcerates and then becomes verrucous with scar formation; often, small black dots are seen in the center and pustulation occurs around the edge. Figure 4 shows a skin lesion characteristic of blastomycosis; 60% of patients have multiple skin lesions. Some patients develop only subcutaneous nodules nearly identical in appearance to the pyogenic subcutaneous abscesses caused by *Staphylococcus aureus*.

Bone involvement is documented in 25-60% of cases. Most often, the vertebrae are the site of infection, but the skull, long bones or ribs may also be affected. Osteomyelitis due to blastomycosis resembles other forms of osteomyelitis in that the patient presents with limitation of motion, pain or swelling overlying the area of involvement. Septic arthritis, usually of the knee, ankle or elbow, can occur when osteomyelitis invades the joint space; a pyogenic synovial fluid is found at arthrocentesis.[16] On X-ray, osteomyelitis due to blastomycosis resembles a pyogenic or tuberculous osteomyelitis.

Involvement of the male genitourinary tract occurs quite frequently and is a telling clinical clue. No other fungal disease endemic in the United States shows this predilection for the male genitourinary tract. The prostate is most often involved, but the epididymis and testis may also be affected. Fluid obtained after prostatic massage can be diagnostic.

Although involvement of the central nervous system is uncommon, it must be recognized promptly since the mortality of this form of blastomycosis is quite high. Typically, the patient develops a chronic meningitis, and analysis of the cerebrospinal fluid yields a lymphocytic pleocytosis, high protein and normal glucose.

Involvement of other organs, including the larynx, has been documented. Of particular note are reports of adrenal involvement, although adrenal insufficiency due to blastomycosis does not occur as often as that due to disseminated histoplasmosis or tuberculosis.

The diagnosis of blastomycosis is established by identifying the yeast in tissue or by culturing the organism in the laboratory.[4,8,12] Potassium hydroxide preparations of appropriate material, such as sputum or prostatic fluid, are occasionally positive

and can show the yeast form as well as the Papanicolaou preparation or sputum cytology can. Figure 5 shows a potassium hydroxide preparation of the sputum of a patient with proven blastomycosis. The organism may on occasion be seen in the sputum gram stain, but in my experience this is a rare occurrence. Nonetheless, use of these techniques can sometimes make bronchoscopy or thoracotomy unnecessary.

The characteristic yeast forms are best seen in tissue specimens stained with methenamine silver or periodic acid-Schiff (PAS) stains, both of which are more sensitive than the routine hematoxylin-eosin stain (Fig. 6). Thermal dimorphism of the cultured organism in the laboratory is a very important clue to the identification of *Blastomyces*. The skin test and serologic procedures are virtually useless in the diagnosis of acute blastomycosis and are more often negative than positive. (In fact, they are more often positive with histoplasmosis than with blastomycosis).[4,17,18]

All cases of blastomycosis should be treated, for it is a progressive disease if unchecked.[4,12] Some authorities question the need for therapy in the acute pneumonic form of the disease.[10] Stilbamidine and amphotericin B have been used for many years to treat blastomycosis, but there are many problems with the toxicity of these agents.[19] As a result, much attention has been given to the new imidazole derivatives, compounds which work by inhibiting ergosterol synthesis and which have few side effects. Clotrimazole has proven to be a useful antifungal agent, but it is limited to topical use because it is absorbed poorly from the gastrointestinal tract. Two other imidazole derivatives, miconazole and ketoconazole, may be used to treat systemic infections. Miconazole has been used to treat blastomycosis, but the drug must be administered

Fig. 4. A typical blastomycotic skin lesion on cheek of 56-year-old male.

intravenously three times daily and the relapse rate is unacceptably high. On the other hand, ketoconazole, a synthetic imidazole, is well absorbed when taken orally and is active in vitro against virtually all fungi, including the dermatophytes.[20] Only mycoses due to *Aspergillus* and *Mucor* species are not consistently susceptible to ketoconazole. There have been few adverse side effects associated with use of this drug; specifically, no significant hematologic or renal abnormalities have been reported. The side effect most frequently reported is nausea, but this can usually be countered by taking the drug with meals. Ketoconazole thus has several major advantages over other antifungal agents: (a) it can

Fig. 5. Note large yeast with wide-based bud in this photomicrograph of potassium hydroxide preparation of expectorated sputum from patient with blastomycosis.

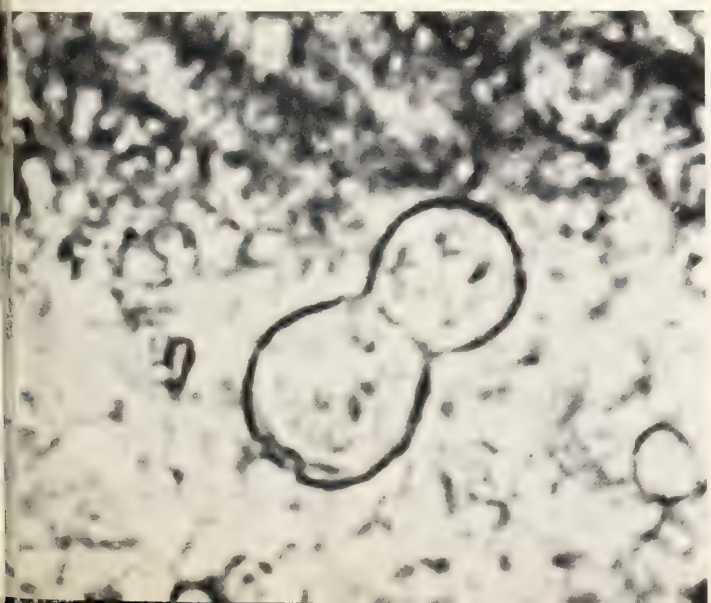
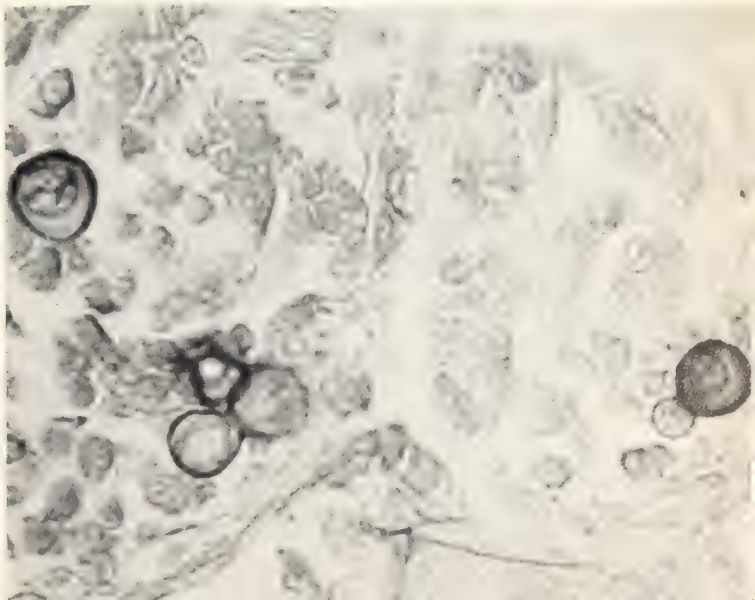


Fig. 6. Photomicrograph of lung biopsy specimen (PAS stain) from patient with blastomycosis showing large yeast forms with wide-based buds.



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be taken in a single oral dose daily; (b) it is well tolerated and causes minimal side effects; and (c) it has a broad spectrum of activity. The drug should have considerable utility in the treatment of diseases which require long therapeutic courses (6-18 months), such as chronic mucocutaneous candidiasis.[21] Clinical trials with ketoconazole have not indicated that resistance develops with prolonged use.

The University of Virginia is one of 14 medical centers nationwide which are conducting clinical trials with ketoconazole. So far, we have enrolled 18 patients with blastomycosis in this study. As of May 1981, three of these patients have been off therapy for six months and are considered cured, 13 have shown improvement and are continuing therapy, and two have not responded to treatment and are considered drug failures.[22] More recently, we have begun enrolling patients with blastomycosis in a protocol comparing the efficacy of 400 mg vs 800 mg daily doses of ketoconazole; the patient discussed today is enrolled in this protocol. Patients with documented histoplasmosis are also eligible for this study.

DR. HOOK: Does person-to-person spread of blastomycosis occur? Should patients with active disease be isolated?

DR. SCHELD: Man-to-man transmission of the fungus by the aerial route is unknown. Patients need not be isolated. I should reemphasize that the so-called "epidemics" of blastomycosis were most likely common-source outbreaks even though the epidemiologic workups were negative.

DR. JACK M. GWALTNEY, JR.: Would you comment on the two patients in your study who did not respond to treatment with ketoconazole?

DR. SCHELD: One patient received a daily dose of 200 mg; we now know that fungicidal serum levels cannot be achieved on such a low dose. The other patient received 400 mg/day, a dose that should have been curative. This patient had skin, lung and bone involvement, and, although the skin and lung disease cleared on therapy, the bone disease continued to progress. Ketoconazole may not be as effective against bone disease as it is against infection in other areas.

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Fragile X-Linked Mental Retardation, a New Entity: Case Reports

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THE fragile X syndrome is a recently recognized cause of moderate to severe mental retardation[1,2]. As this disorder segregates in families like a single gene mutation on the X chromosome and as it occurs with relatively high frequency, it represents one of the most common opportunities for the prevention of mental retardation. We cite our experience with this condition to alert Virginia physicians to its existence and the means for establishing a specific diagnosis.

This disorder segregates in families like hemophilia, color blindness and Duchenne muscular dystrophy, i.e., through carrier females and affected males. Affected males do not usually manifest any physical features which suggest the diagnosis before puberty. After puberty, the diagnosis is suggested by large testes, large and simple ears, and a "jocular" personality[3]. The diagnosis is confirmed by the demonstration of the "fragile X chromosome" in cultured lymphocytes. This demonstration requires the use of special culture media. The fragile X is so designated because the tips of the long arm of the X chromosome appear to have broken off or to be attached by a thin thread[4].

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Case Reports

Case 1

This 15-year-old male was the 3850-gm product of a normal pregnancy and delivery to a then 29-year-old G_3 woman. His childhood health was normal, but retarded intellectual development and hyperactivity were apparent in late infancy. Because of significant retardation and hyperactive behavior, he was admitted to the Lynchburg Training School and Hospital at 12 years of age. At 15 years of age he was moved to a home for retarded teenagers.

His parents were normal and unrelated. He had a normal older brother and sister, a maternal cousin who was retarded secondary to perinatal asphyxia, and a paternal cousin with a dysmorphic retardation syndrome and normal karyotype.

Physical exam revealed an attractive, cooperative male who appeared younger than his 15 years. Height, weight and head circumference were age appropriate. The only abnormalities noted on exam were large, simple ears and macroorchidism; the left testis was 6.0 by 4.5 cm and the right testis was 5.5 by 4.5 cm (mean testicular length in adult males is 4.5-5.0 cm). Testing by the Peabody Picture Vocabulary Test and the Stanford-Binet revealed an IQ of approximately 20.

He was described by care providers as moody with periods of tantrums, aggressive behavior with short attention span, periods of passivity with little personal interaction, and periods of agreeableness, playfulness and cooperation. A program of behavior modification has yielded inconsistent results.

Lymphocytes cultured in GIBCO medium 199 with added methotrexate showed the fragile X chromosome to be present in 31% of the cells examined. Similar chromosome studies on his mother and sister disclosed no cells bearing the fragile X chromosome. The sister underwent amniocentesis with a subsequent pregnancy which revealed a 46,XX karyotype and later delivered a normal female infant.

Cases 2 and 3

These brothers, aged 44 and 37 years, were seen

at the Lynchburg Training School and Hospital for evaluation following a request for genetic counseling by their brother. Both were products of normal term pregnancies, with birth weights of 4100 and 2950 gms respectively. Their general health was good, but retarded development was noted from early infancy. They were described as rambunctious and stubborn. Their admission to the Lynchburg Training School and Hospital in 1949 was at 12 and 5 years of age.

They were the second and fourth born of five brothers. The first born, by a different father, had been admitted to the same facility several years before his brothers and died there of unknown causes. The third and fifth born brothers were normal adults, aged 40 and 34 years. There was no other family history of mental retardation. The boys' mother had no brothers.

The physical examination of both brothers was generally normal except for large, simple ears, 7.5 cm bilaterally in one and 8.2 cm bilaterally in the other (6.0 cm is mean ear length and 7.5 cm is at the 97th percentile for adults); a happy, jocular personality and macroorchidism (6.5 × 5.5 cm bilaterally in one brother and 6.0 × 5.8 cm bilaterally in the other).

Psychometric evaluation by the Peabody Picture Vocabulary Test and the Slosson Intelligence Test gave estimated IQs of 20 for each brother. Neither exhibited evidence of an affective disorder and both had good attention spans. They had excellent self-help skills and were cooperative in group activities.

Chromosome studies of cultured lymphocytes as described above revealed 8% and 9% of cells with the fragile X chromosome in the two brothers.

Discussion

Cytogenetic Studies

In 1969 Lubs described the presence of a morphologically distinct ("marker") X chromosome in retarded males of a family whose pedigree suggested X-linked recessive inheritance[5]. The fragile X chromosome (Fig. 1) had what appeared to be a gap or thread-like elongation at a consistent location (Xq27-28). The tips of the long arm of the X chromosome appeared like two rounded satellites. There were no further reports until that of Harvey et al in 1977[1]. Since that report, numerous families with this syndrome have been described.

It is generally agreed that the fragile X chromosome is a specific cellular marker of a form of X-linked mental retardation characterized by the presence of macroorchidism in post-adolescent males. The biochemical basis for the expression of the marker or the role of the fragile site in the expression of this syndrome is unknown.

Clinical Phenotype

It has long been recognized that moderate to profound mental retardation occurs with an unequal sex distribution, males being more commonly affected than females. With single gene mutations on the X chromosome, the double X bearing female with such a mutation is usually a phenotypically normal female carrier or when affected, shows milder clinical features than an affected male. Thus, numerous X-linked recessive disorders could in large part account for the altered sex ratio in mental retardation[6-8]. Examples include the Hunter syndrome (mucopolysaccharidosis II), Menke syndrome (kinky hair syndrome), Lowe syndrome (oculocerebro-renal syndrome), Lesch-Nyhan syndrome (hyperuricemia), and Duchenne muscular dystrophy. Sex chromosome abnormalities also contribute to the altered sex ratio. The Klinefelter syndrome, 47,XXY, is common at one in 1,000 male infants, and mild to moderate mental retardation is not uncommon. The Turner syndrome, 45,X, is less frequent at one in 5,000 female infants, and mental

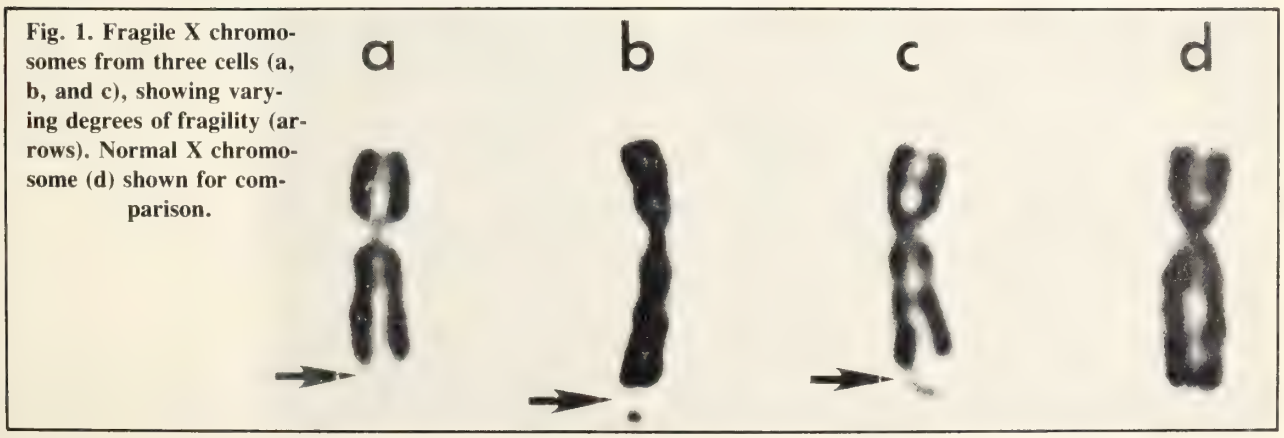

Fig. 1. Fragile X chromosomes from three cells (a, b, and c), showing varying degrees of fragility (arrows). Normal X chromosome (d) shown for comparison.

retardation usually is not a feature. The fragile X syndrome, however, is the most common disorder contributing to the altered sex ratio.

It has been appreciated for many years that there were families in which "non-specific mental retardation" occurred in males in a pattern consistent with X-linked recessive inheritance[6]. Subsequently, macroorchidism was recognized as a consistent feature in some of these pedigrees[3]. With the advent of a specific cellular marker, the fragile X syndrome has emerged as a common disorder, estimated to occur in one in 1,000 live-born male infants. Heterozygote females for the fragile X syndrome do not totally escape the consequences of the mutation. Approximately 10% of female carriers demonstrate mild mental retardation. With one in 500 females being heterozygous for the X-linked mutation and 10% of them retarded, the incidence of mental retardation in females is one in 5,000[9]. Thus approximately 45 infants are born each year in Virginia with this form of mental retardation.

Routine chromosome studies will not reveal the fragile X chromosome which is the cellular marker of this mental retardation syndrome. There are no consistent physical findings in preadolescent retarded males or in mildly retarded females to suggest a clinical diagnosis. Special culture media using the pyrimidine analogue FudR5-fluorodeoxyuridine is required to demonstrate the fragile X. The diagnosis of an affected individual, male or female, may identify a family with numerous, phenotypically normal females who are carriers with a 25% risk of having a severely retarded son and a 2.5-3.0% risk of having a mildly retarded daughter.

There are currently two major problems in attempts to prevent mental retardation due to the fragile X syndrome. First, the fragile X chromosome cannot be demonstrated in cultured amniotic fluid cells when there is an affected male fetus. There are preliminary reports which suggest this should be possible in the near future[10]. Until such is available, a female at high risk may elect an amniocentesis with the intent of continuing the pregnancy with a female fetus; such occurred in Case 1 as cited. The second problem is more difficult to resolve. A family at risk is identified only after the diagnosis in an index case. If the condition is recognized only by the presence of macroorchidism in retarded, post-adolescent males, then numerous other pregnancies may have already occurred in the family before the risks were appreciated. To overcome this problem, a high index of suspicion is required and an aggressive use of fragile X chromosome studies is necessary.

In order to increase the ascertainment rate and, therefore, provide accurate genetic counseling, it is recommended that blood samples be obtained specifically for fragile X chromosome analysis when:

1. A post-adolescent retarded male is found to have macroorchidism.

2. The family history is suggestive of an X-linked form of mental retardation, i.e., affected brothers, affected nephew and maternal uncle, or a mildly retarded female with a significantly retarded brother or other maternally related male relative. (Macroorchidism has not been found in a small number of families with X-linked mental retardation and the fragile X syndrome. Thus a pedigree suggesting non-specific X-linked mental retardation should warrant study even in the absence of macroorchidism. Because of the low incidence and small percentage of cells with the fragile X chromosome, studies are not currently recommended in isolated instances of mild mental retardation in females).

3. A pre-adolescent, retarded male has a generally normal physical exam and no physical or laboratory findings establishing a specific cause of the retardation.

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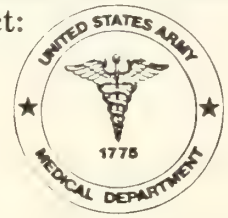

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WARNINGS: ASPIRIN: Use salicylates with extreme caution in patients with peptic ulcer, asthma, coagulation abnormalities, hypoprothrombinemia, vitamin K deficiency, or those on anticoagulants. In rare instances, aspirin in persons allergic to salicylates may result in life-threatening allergic episodes.

MEPROBAMATE: DRUG DEPENDENCE: Physical and psychological dependence, and abuse have occurred. Chronic intoxication from prolonged ingestion of, usually, greater than recommended doses is manifested by ataxia, slurred speech, and vertigo. Therefore, carefully supervise dose and amounts prescribed and avoid prolonged use, especially in alcoholics and others with known propensity for taking excessive quantities of drugs. Sudden withdrawal after prolonged and excessive use may precipitate recurrence of preexisting symptoms, e.g. anxiety, anorexia, or insomnia, or withdrawal reactions, e.g., vomiting, ataxia, tremors, muscle twitching, confusional states, hallucinosis, and, rarely, convulsive seizures. Such seizures are more likely in persons with CNS damage or preexistent or latent convulsive disorders. Onset of withdrawal symptoms occurs usually within 12 to 48 hours after discontinuation; symptoms usually cease within next 12-to-48-hour period. When excessive dosage has continued for weeks or months, reduce dosage gradually over 1 to 2 weeks rather than stop abruptly. Alternatively, a short-acting barbiturate may be substituted, then gradually withdrawn.

POTENTIALLY HAZARDOUS TASKS: Warn patients meprobamate may impair mental or physical abilities required for potentially hazardous tasks, e.g., driving or operating machinery.

ADDITIVE EFFECTS: Since CNS-suppressant effects of meprobamate and alcohol or meprobamate and other psychotropic drugs may be additive, exercise caution with patients taking more than one of these agents simultaneously.

USAGE IN PREGNANCY AND LACTATION: An increased risk of congenital malformations associated with minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during first trimester of pregnancy, has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at time of institution of therapy should be considered. Advise patients if they become pregnant during therapy or intend to become pregnant to communicate with their physicians about desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breastfeeding patients, consider the drug's higher concentrations in breast milk as compared to maternal plasma levels.

USAGE IN CHILDREN: Keep preparations with aspirin out of reach of children. Equagesic®-M is not recommended for patients 12 years of age and under.

PRECAUTIONS: ASPIRIN: Salicylates antagonize uricosuric activity of probenecid and sulfinpyrazone. Salicylates are reported to enhance hypoglycemic effect of sulfonylurea antidiabetics.

MEPROBAMATE: Use lowest effective dose, particularly in elderly and/or debilitated, to preclude over-sedation. Meprobamate is metabolized in the liver and excreted by the kidney, to avoid excess accumulation exercise caution in its use in patients with compromised liver or kidney function. Meprobamate occasionally may precipitate seizures in epileptic patients. It should be prescribed cautiously and in small quantities to patients with suicidal tendencies.

ADVERSE REACTIONS: ASPIRIN: May cause epigastric discomfort, nausea and vomiting. Hypersensitivity reactions, including urticaria, angioneurotic edema, purpura, asthma, and anaphylaxis may rarely occur. Patients receiving large doses of salicylates may develop tinnitus.

MEPROBAMATE: CNS: Drowsiness, ataxia, dizziness, slurred speech, headache, vertigo, weakness, paresthesias, impairment of visual accommodation, euphoria, overstimulation, paradoxical excitement, fast EEG activity.

GI: Nausea, vomiting, diarrhea.

CARDIOVASCULAR: Palpitation, tachycardia, various forms of arrhythmia, transient ECG changes, syncope, hypotensive crisis.

ALLERGIC OR IDIOSYNCRATIC: Milder reactions are characterized by itchy, urticarial, or erythematous maculopapular rash, generalized or confined to the groin. Other reactions include leukopenia, acute nonthrombocytopenic purpura, petechiae, ecchymoses, eosinophilia, peripheral edema, adenopathy, fever, fixed drug eruption with cross-reaction to carisoprodol, and cross-sensitivity between meprobamate/mebutamate and meprobamate/carbromal. Rare, more severe hypersensitivity reactions include hyperpyrexia, chills, angioneurotic edema, bronchospasm, oliguria, and anuria. Also, anaphylaxis, exfoliative dermatitis, stomatitis, and proctitis. Stevens-Johnson syndrome and bullous dermatitis have occurred.

HEMATOLOGIC (SEE ALSO "ALLERGIC OR IDIOSYNCRATIC"): Agranulocytosis, aplastic anemia have been reported, although no causal relationship has been established, and thrombocytopenic purpura.

OTHER: Exacerbation of porphyric symptoms.

DOSAGE AND ADMINISTRATION: Usual dose is one or two tablets, 3 to 4 times daily as needed for relief of pain when tension or anxiety is present. Not recommended for patients 12 years of age and under.

OVERDOSAGE: Treatment is essentially symptomatic and supportive. Any drug remaining in the stomach should be removed. Induction of vomiting or gastric lavage may be indicated. Activated charcoal may reduce absorption of both aspirin and meprobamate. Aspirin overdosage produces usual symptoms and signs of salicylate intoxication. Observation and treatment should include management of hyperthermia, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions. Suicidal attempts with meprobamate have resulted in drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse. Some suicidal attempts have been fatal. The following data, reported in the literature and from other sources, are not expected to correlate with each case (considering factors such as individual susceptibility and length of time from ingestion to treatment), but represent usual ranges reported. Acute simple overdose (meprobamate alone): Death has been reported with ingestion of as little as 12 gram meprobamate and survival with as much as 40 gram.

BLOOD LEVELS:

0.5-2.0 mg percent represents usual blood-level range after therapeutic doses. The level may occasionally be as high as 3.0 mg percent.

3-10 mg percent usually corresponds to findings of mild-to-moderate symptoms of overdosage, such as stupor or light coma.

10-20 mg percent usually corresponds to deeper coma, requiring more intensive treatment. Some fatalities occur.

At levels greater than 20 mg percent more fatalities than survivals can be expected.

Acute combined overdose (meprobamate with other psychotropic drugs or alcohol): Since effects can be additive, history of ingestion of a low dose of meprobamate plus any of these compounds (or of a relatively low blood or tissue level) cannot be used as a prognostic indicator.

In cases of excessive doses, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Any drug remaining in stomach should be removed and symptomatic treatment given. Should respiration or blood pressure become compromised, respiratory assistance, CNS stimulants and pressor agents should be administered cautiously as indicated. Diuresis, osmotic (mannitol) diuresis, peritoneal dialysis, and hemodialysis have been used successfully in removing both aspirin and meprobamate. Alkalinization of the urine increases excretion of salicylates. Careful monitoring of urinary output is necessary, and caution should be taken to avoid overhydration.

Relapse and death, after initial recovery, have been attributed to incomplete gastric emptying and delayed absorption.

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Pneumonia Due to Eikenella Corrodens: Case Report

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Sharon E. Oster, MD,
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Newport News, Virginia

REPORTS have implicated *Eikenella corrodens*, a fastidious aerobic gram-negative rod, in many types of infections, such as meningitis,[1,2] skin abscess,[1,2,3] endocarditis[1] and pneumonia.[5,6] In all previous reports of pulmonary infections in adults, *E corrodens* has been recovered as part of a mixed culture.[6] We here report a case in which this organism was isolated in pure culture, demonstrating an ability to act alone in causing pneumonia.

Case Report

A 46-year-old man was admitted to Riverside Hospital, Newport News, Virginia, on 5/27/81. His symptoms included a one-month history of production of copious foul-tasting sputum, dyspnea, cough and low-grade fever. The patient had a 48-pack-year smoking history but claimed to have reduced cigarette use to only a few each day after being diagnosed as having bronchiectasis two years previously. He had been treated with trimethoprim/sulfamethoxazole, 2 tablets po bid, for three weeks prior to admission. Two days before admission this drug was discontinued and amoxicillin 250 mg po tid was substituted.

On initial physical examination the patient was an ill-appearing, middle-aged male with a fever of 39°C and respiratory rate of 30/min. The examination was normal except for chest findings, which revealed bibasilar dry rales more pronounced on the right. He was coughing up foul-smelling, greenish sputum.

Admission laboratory data revealed a WBC of 15,000/mm^3, with 53% segmented neutrophiles, 19% bands, 8% monocytes, 19% lymphocytes and 1% eosinophiles. The hemoglobin was normal (14.5 gm%) as were routine chemistry studies. The chest X-ray demonstrated patchy infiltrates in both lung bases as well as the lingula and left upper lobe. The radiologist reported changes caused by emphysema and bronchiectasis at both lung bases as in previous X-rays. A Gram's stain of expectorated sputum showed many polymorphonuclear leukocytes as well as occasional gram-positive cocci and gram-negative rods. The sputum culture was reported as normal flora.

Cefamandole 2 gram IV q6h was started on 5/27/81 along with postural drainage. The patient continued to produce copious amounts of purulent sputum and spike fevers daily to 40°C. Repeat chest X-rays on 6/1/81 and 6/6/81 showed progressively increasing infiltrates in the left mid-lung field.

On 6/8/81 an infectious disease consultant was asked to see the patient. Consequently, cefamandole was discontinued and sputum cultures and smears were again obtained. Routine aerobic bacterial sputum cultures, AFB stain, potassium hydroxide preparation and sputum cytology were all non-diagnostic. The Gram's stain still revealed abundant PMNs with only few bacteria. A PPD skin test was negative. Acute (and later convalescent) indirect fluorescent antibody titers for Legionella species were obtained and were negative.

Fiberoptic bronchoscopy was performed through an endotracheal tube on 6/10/81 at which time purulent secretions, erythema and mucosal edema were noted extending from the trachea to both mainstem bronchi. Bronchial brushings and washings were obtained and were negative for malignancy. Bronchoscopy specimens were also submitted for fungus, AFB, aerobic (including charcoal yeast extract agar for Legionella species) and anaerobic cultures as well as Gram's stain, AFB and fungal smears. Many thin gram-negative rods were observed on Gram's stain in addition to abundant PMNs. Fungal and AFB smears were negative.

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Submitted 3-23-82.

Immediately after bronchoscopy, cefamandole was restarted (2 gm IV q4h) as well as tobramycin (100 mg IV q8h). The fever remained unchanged and on 6/12/81 cefamandole was discontinued and erythromycin (750 mg IV q6h) begun. The fever decreased, and by 6/15/81 the patient had become afebrile and had improved clinically. Oral erythromycin (500 mg q6h) was continued and tobramycin discontinued in the belief that the former drug was responsible for clinical improvement. The patient did well, his sputum production decreased, and he was discharged on 6/19/81. He continued to take erythromycin for two more weeks and recovery was uneventful.

On 6/17/81 a pure culture of a gram-negative rod was isolated from bronchoscopy specimens and tentatively identified as *Eikenella corrodens*. The organism was forwarded to the Virginia State Bacteriology Laboratory, Richmond, Virginia, which confirmed the identification. Due to its fastidious nature, routine antimicrobial sensitivity tests could not be performed. The organism was sent to the Centers for Disease Control, Special Pathogens Branch, where Mueller-Hinton broth dilution MIC testing was performed. The following results were obtained: amikacin 16 μg/ml, ampicillin 0.25 μg/ml, carbenicillin ≤ 4 μg/ml, cefamandole 2 μg/ml, cefoxitin 0.5 μg/ml, cephalothin 8 μg/ml, chloramphenicol 1 μg/ml, erythromycin 4 μg/ml, gentamicin 4 μg/ml, penicillin 0.5 μg/ml, trimethoprim/sulfamethoxazole ≤ 0.6 μg/ml/0.03 mg/ml, tetracycline 0.5 μg/ml, tobramycin 4 μg/ml, vancomycin > 16 μg/ml, oxacillin > 8 μg/ml, clindamycin > 8.0 μg/ml.

Discussion

Eikenella corrodens was first described in 1958 by Eiken, who named the organism *Bacteroides corrodens* because it grew anaerobically and produced pitting colonies on agar plates after 36-48 hours growth.[7] In 1971 the organism was renamed *Eikenella corrodens* when it was found that it would also grow well aerobically, depending on the hemin content of the agar.[8] Homology studies further demonstrated that this facultative organism differed from strictly anaerobic gram-negative rods in G + C content of DNA and was identical to another group of organisms designated HB-1.[8]

E corrodens is a normal inhabitant of the oral mucosa and has frequently been reported in soft tissue and abdominal wound infections.[1,3] The distinguishing features of many of these infections are their indolent nature, association with other bacteria, history of oral or abdominal contamination and production of a foul odor; in many ways they mimick anaerobic infections. Less commonly, *E corrodens* has been isolated alone in osteomyelitis,[4] central nervous system infections[1,2] arthritis,[9] endocarditis[1] and empyema,[5] the last case occurring in a 6-month-old child with pneumonia in whom the organism was recovered by thoracentesis.

The largest series of pulmonary infections involving *E corrodens* was reported by Goldstein et al,[6] who described seven cases, all adults, in whom this organism was recovered either by transtracheal aspiration or percutaneous lung aspiration. In none of these patients was *E corrodens* the sole pathogen. Several patients failed to respond to therapy with a penicillinase-resistant penicillin, clindamycin or a cephalosporin.

Our present case in many ways was characteristic of previously described *E corrodens* pulmonary infections in that the course was indolent, progressive and unresponsive to routine broad-spectrum antimicrobial coverage with a cephalosporin and an aminoglycoside. The patient responded promptly and dramatically when treated with erythromycin even before the organism was identified and sensitivity results known. Erythromycin was chosen empirically due to the consideration of either Legionellosis or anaerobic pneumonia since in vitro studies also indicate efficacy against the latter group of pathogens.[10] As has been shown by others,[1,3] disc sensitivity testing of *E corrodens* is unreliable and usually requires agar or broth dilution MIC analysis.

This case report establishes the ability of *E corrodens* to act alone in causing pneumonia. This organism mimicked an anaerobic pulmonary infection in that it produced a pneumonia with foul-smelling sputum. Although previous in vitro data reveals that penicillin, cefoxitin and tetracycline have the most favorable MICs,[6,7] *E corrodens* is uniformly resistant to clindamycin and metronidazole.[3] Several studies have demonstrated only variable sensitivity to cephalosporins.[1,3,6] An interesting finding in our case was that although the MIC to cefamandole showed the organism to be quite sensitive in vitro (2 μg/ml), the in vivo experience was quite the opposite. It is also interesting to note that this patient did not respond to amoxicillin and trimethoprim/sulfamethoxazole despite good in vitro sensitivity results. Erythromycin, on the other hand, with only intermediate sensitivity in vitro (4 μg/ml), was an effective therapeutic agent.

A possible explanation for the discrepancy between the patient's response to antibiotic therapy and the sensitivities of the organism obtained at

bronchoscopy might be an "inoculum effect." As has been shown with other bacteria, a relatively small concentration of microorganisms may appear sensitive to a drug whereas a larger concentration demonstrates more resistance (higher MICs). The inoculum effect was not examined in this case. Another possibility might simply be that other organisms may have been present which were resistant to the antibiotics used prior to initiation of erythromycin. We cannot prove or disprove this point except to indicate that a meticulous effort was made with the means available to recover the organism(s) responsible for this infection.

From reviewing the literature and experience with our case, we would conclude that *E corrodens*, either alone or in combination with other bacteria, may be responsible for serious progressive pulmonary infections and should be considered in patients not responding to cephalosporin and aminoglycoside antimicrobial coverage. As in other types of atypical pneumonias, such as mycoplasma or Legionellosis, erythromycin may prove to be among the drugs of choice.

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ABSTRACTS

These are abstracts of papers to be presented at the annual meeting of the Virginia Surgical Society from April 29 to May 1 in Williamsburg. Stanton P. Nolan, MD, is program chairman.

Traumatic Diaphragmatic Injuries. Joseph T. Mullen, MD, *Norfolk.*

Diaphragmatic injuries are not always promptly recognized. In this report experience is reviewed with diaphragmatic injuries in 42 patients, 22 of whom were treated during a five-year period before July 1973 at Naval Hospital, Portsmouth, and 20 during a six-year period prior to July 1979 at Norfolk General Hospital.

Diaphragmatic injury as a single entity was rare, whether the injuring agent was blunt or penetrating. In several instances operative management was delayed because of delay in diagnosis. A high index of suspicion, careful examination of chest roentgenogram and appropriate barium contract studies should make for early diagnosis.

Improved Survival Following Complete Excision of Retroperitoneal Sarcomas. Patrick C. McGrath, MD, James P. Neifeld, MD, Walter Lawrence, Jr., MD, Richard M. DeMay, MD, J. Shelton Horsley III, MD, and George A. Parker, MD, *Richmond.*

Retroperitoneal sarcomas have been associated with a high local recurrence rate and a poor prognosis; total resection may be difficult due to proximity to vital structures. Management of these tumors at our institution has been reviewed to assess extent of surgical resection and the addition of other therapy on survival.

Charts and slides of 45 patients (1964-1982) with primary retroperitoneal sarcoma (excluding pediatric rhabdomyosarcoma) were reviewed to determine clinical presentation, histologic type and grade, extent of surgical resection, operative morbidity and mortality, use of radiation and/or chemotherapy, and survival data. The majority of patients presented with pain and a palpable mass. There were 13 leiomyosarcomas, 12 liposarcomas, 6 malignant fibrous histiocytomas and 14 miscellaneous tumors.

Seventeen of the 45 patients (38%) had complete tumor excision, 66% required resection of adjacent organs. These patients underwent 21 operations with a morbidity of 33% and no operative mortality. The median disease-free 5-year survival was 65% with an overall 70% survival at 5 years; corresponding 10 year disease-free survival was 25% with an overall 58% survival. Eleven patients (65%) developed recurrent disease at a mean interval of 5 years following complete excision. Only 6 patients received adjuvant radiation and/or chemotherapy with 4 remaining disease-free for 46 to 61 months.

Seventeen patients underwent partial excision of tumor and 11 patients underwent biopsy only; they had similar survival curves with only 4% alive at five years. These 28 patients underwent 59 operative procedures with a morbidity of 17% and an operative mortality of 5%; median time to clinical evidence of tumor progression was 12 months. Sixty percent of these patients received therapeutic radiation and/or chemotherapy, but their survival was the same as those undergoing surgery alone.

These data emphasize the importance of an aggressive surgical approach in the treatment of retroperitoneal sarcomas. Complete tumor resection and total excision of recurrences will allow many patients long-term survival.

Lipolysis, a New Technique for Removing Body Fat. Richard A. Mladick, MD, and Richard L. Morris, MD, *Virginia Beach.*

Lipolysis, a technique developed in 1977 by Dr. Illouz of Paris, has recently been introduced in the United States. The author (R.A.M.) learned the technique from Dr. Illouz and will present his experience with removal of body fat from hips, abdomen, knees, chin, etc. The technique is performed thru a ½-inch incision. Up to 4 pounds of body fat can be removed by this technique, which employs a very strong suction machine.

Vascular Leiomyoma of the Superior Mesenteric Vein. John T. Mazzeo, MD, *Woodbridge.*

Vascular leiomyomas as benign tumors arising from the smooth muscle of major veins are extremely rare, with only ten cases reported. The first reported vascular leiomyoma arising from the superior mesenteric vein is described. The pathologic and angiographic criteria used in its diagnosis and preoperative evaluation are considered.

Identification of the tumor originating from the smooth muscle, although ideal, is not always possi-

ble and has been identified in only two cases. The diagnosis also depends on separating the vascular leiomyomas from other benign venous tumors. The ratio of smooth muscle elements to vascular channels is higher in the leiomyoma and reversed in the primarily hemangioma-type tumors. Differentiation of truly benign tumors from low-grade invasive and highly malignant tumors can be difficult.

Tumors which have less than 20 mitoses per hpf showed no recurrent of tumor after surgery. However, this may not always be true and should be used only as a guide. Therefore, wide surgical extirpation of the tumor mass and adjacent vessels and periconnective tissue is the treatment of choice. Preoperative evaluation via phlebography and, in this case, venous phase arteriography should be used in planning the surgical approach.

Techniques for Reduction of Spinal Cord Complications in Surgery for Thoracoabdominal Aneurysms. I. K. Crosby, MD, I. L. Kron, MD, and R. M. Mentzer, MD, *Charlottesville.*

Spinal cord complications following resection of thoracoabdominal aneurysms are seen in as many as 23% of patients as reported by different authors. Improvements in anesthetic and surgical techniques can reduce the incidence of these spinal cord complications.

Eight patients presented to the University of Virginia from May through December 1982 with symptomatic thoracoabdominal aneurysms (25% were ruptured at presentation). The ages of the patients ranged from 56 to 77 years (average 66 years) and 50% of the patients had undergone a prior infrarenal abdominal aortic aneurysm resection. Anesthetic techniques to minimize "complications" included a) Swan Ganz catheter monitoring intraoperatively; and b) afterload reduction with nitroprusside to maximize cardiac output (especially during the period of aortic cross-clamping). Surgical techniques included a) bevelling of the proximal anastomosis to preserve intercostal vessels; b) bevelling of the distal anastomosis to preserve lumbar vessels; c) reimplantation of the intercostal vessels into the tubular prosthetic graft; d) preoperative planning to reduce the cross-clamp time; and e) stabilization of the cardiac output in the postoperative phase. One 74-year-old man with significant cardiac disease died of left ventricular failure early in the postoperative phase, but the other seven patients survived with no evidence of spinal cord complications.

The simplified revascularization with the inlying prosthetic tube graft and the reimplantation of visceral vessels is technically attractive, and attention to anesthethetic and surgical techniques can minimize spinal cord complications.

Pseudolymphoma of the Stomach, a Challenging Clinical Problem. Richard K. Orr, MD, John R. Lininger, MD, and Walter Lawrence, Jr., MD, *Richmond.*

Pseudolymphoma of the stomach is a benign inflammatory condition associated with chronic gastric ulcer, but it mimics carcinoma and malignant lymphoma. The experience with this disorder at the Medical College of Virginia Hospitals was reviewed to assess our approach to clinical and pathologic diagnosis and to treatment.

There were eight patients with the ultimate diagnosis of gastric pseudolymphoma in the years 1969 to 1982, all patients being treated by operation. Six presented with symptoms and with radiographic and endoscopic findings consistent with chronic gastric ulcer. All of these patients had symptoms of more than six months duration, had failed medical management, and were considered operative candidates on the basis of their chronic symptoms. Two additional patients presented with shorter histories of similar symptoms but required emergency operative management for perforation and bleeding, respectively.

The dilemma produced by most of the patients in this series was the differentiation of this unusual inflammatory process from a malignant lesion since the extent of surgery considered appropriate would differ according to the diagnosis. Pathologic assessment by preoperative endoscopic biopsy and operative biopsy proved difficult. The characteristic histologic findings on the resected lesions were follicle formation with true germinal centers, a mixed inflammatory infiltrate, and no involvement of regional lymph nodes. Recently, staining the biopsy material with immunoperoxidase has been utilized for differentiation of this lesion from malignant lymphoma.

All eight patients in this series underwent partial gastrectomy of varying degree but the two presenting with acute problems had more extensive resection due to a persistent clinical suspicion of carcinoma or malignant lymphoma. Patient followup revealed no subsequent clinical problems, except for one patient who presented with diffuse Hodgkin's disease 30 months after operation despite confidence that the resected lesion was pseudolymphoma.

This series and a review of the literature will be presented.

Vertebral-to-Carotid Artery Transposition, a Surgical Treatment for Vertebral Basilar Insufficiency. Stanley O. Snyder, Jr., MD, Roger T. Gregory, MD, Robert G. Gayle, MD, Jock R. Wheeler, MD, and Ralph B. Pfeiffer, Jr., MD, *Norfolk.*

The symptoms of vertebral basilar insufficiency are often vague and nonspecific. Traditionally, treatment has been almost entirely nonoperative. However, there exists a group of patients with significant proximal vertebral artery stenoses whose vertebral basilar insufficiency symptoms can be alleviated by surgical correction of these lesions.

Fourteen patients underwent vertebral–to–common–cartoid artery transposition over a two-year period for symptoms of severe vertebral basilar insufficiency. All patients showed severe stenosis of the operated vertebral, while the contralateral vertebral was either absent, hypoplastic or stenotic. Nine patients were completely relieved of these symptoms, four improved, and one remained unchanged, during an average followup period of 15 months. The preoperative assessment, surgical technique and review of the literature is included.

Foreign Bodies in the Airways of Children. Bradley M. Rodgers, MD, *Charlottesville.*

Foreign body aspiration is responsible for approximately 2000 deaths a year in children under the age of 6 years. In most children the aspiration is manifested by the triad of choking while eating, coughing and wheezing, but the presentation may be considerably more subtle, resulting in prolonged and misguided therapy.

A review of airway foreign bodies encountered in children at the University of Virginia between 1970 and 1980 has been conducted. Approximately 20% of these children did not have a definite history of aspiration at the time of initial encounter with the physician. The most frequent physical finding encountered was localized wheezing, but a prefectly normal physical examination was described not infrequently. Chest roentgenograms on inspiration and expiration were the most useful examination in establishing the diagnosis. Only a rare foreign body was radiopaque.

The decision to perform endoscopy was based on a history suggestive of aspiration with or without positive physical or radiologic findings. In many of these patients this decision was delayed because of a failure to have a high index of suspicion in these patients. Experience with endoscopy in such patients over this interval has lead to several specific recommendations for anesthetic and surgical management.

Acute Cecal Herniation Through the Foramen of Winslow. Amiele H. Barakey, MD, *Chesapeake.*

Plain X-rays of the abdomen correlated with suggestive history and revealing physical exam prompted a correct preoperative diagnosis of acute herniation through the foramen of Winslow. Although recent literature has expounded contrast X-ray studies suggestive or diagnostic of herniation through the foramen of Winslow, it is believed that in a significant number of cases the correct interpretation of plain abdominal films will lead to early corrective surgery. Corrective surgical treatment of an acutely incarcerated cecum through the foramen is discussed.

Reconstruction of Massive, Full-Thickness, Abdominal Wall Defect. A. I. Mehrhof, Jr., MD, S. Dawson Theogaraj, MD, and I. Kelman Cohen, MD, *Richmond.*

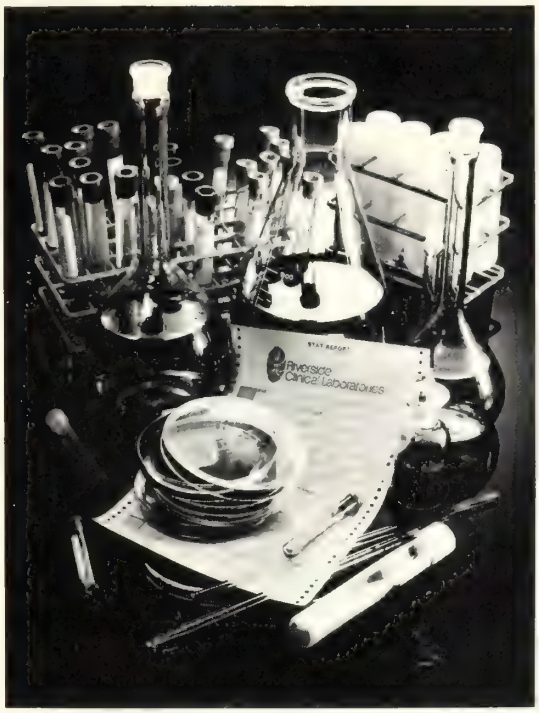
Reconstruction of a massive, full-thickness abdominal defect secondary to an electrical burn injury utilizing both an extended tensor fascia lata musculocutaneous flap and a groin flap will be demonstrated. Preoperative views and a complete step by step videotape will clearly demonstrate the techniques for massive abdominal wall reconstruction. Anatomy is illustrated and potential pitfalls explained. Followup of the result of the reconstruction will also be provided.

A Modification in Technique for Mesocaval Shunt. Robert W. Knapp, MD, *Portsmouth.*

Despite advancements in supportive care, there remains an indication in some patients for acute portosystemic decompression to stop exsanguinating hemorrhage from esophageal varices. The benchmark for such procedures is the side-to-side portocaval shunt.

Side-to-side shunting may be a formidable task in patients who have had upper abdominal surgery, or when hepatic size or available assistant forces limit exposure. Interposition "H" mesocaval shunt may be a viable alternative for those instances. Primary among criticisms of a prosthetic mesocaval interposition has been an alleged high occlusion/rebleed rate. The author feels his modification in the "H" graft helps eliminate kinking, a central cause of shunt occlusion. A wedge modification in the prosthesis has been employed since 1974 with apparent satisfactory patency rate.

The author's last 12 cases are outlined, and the technique of graft modification and placement are described.



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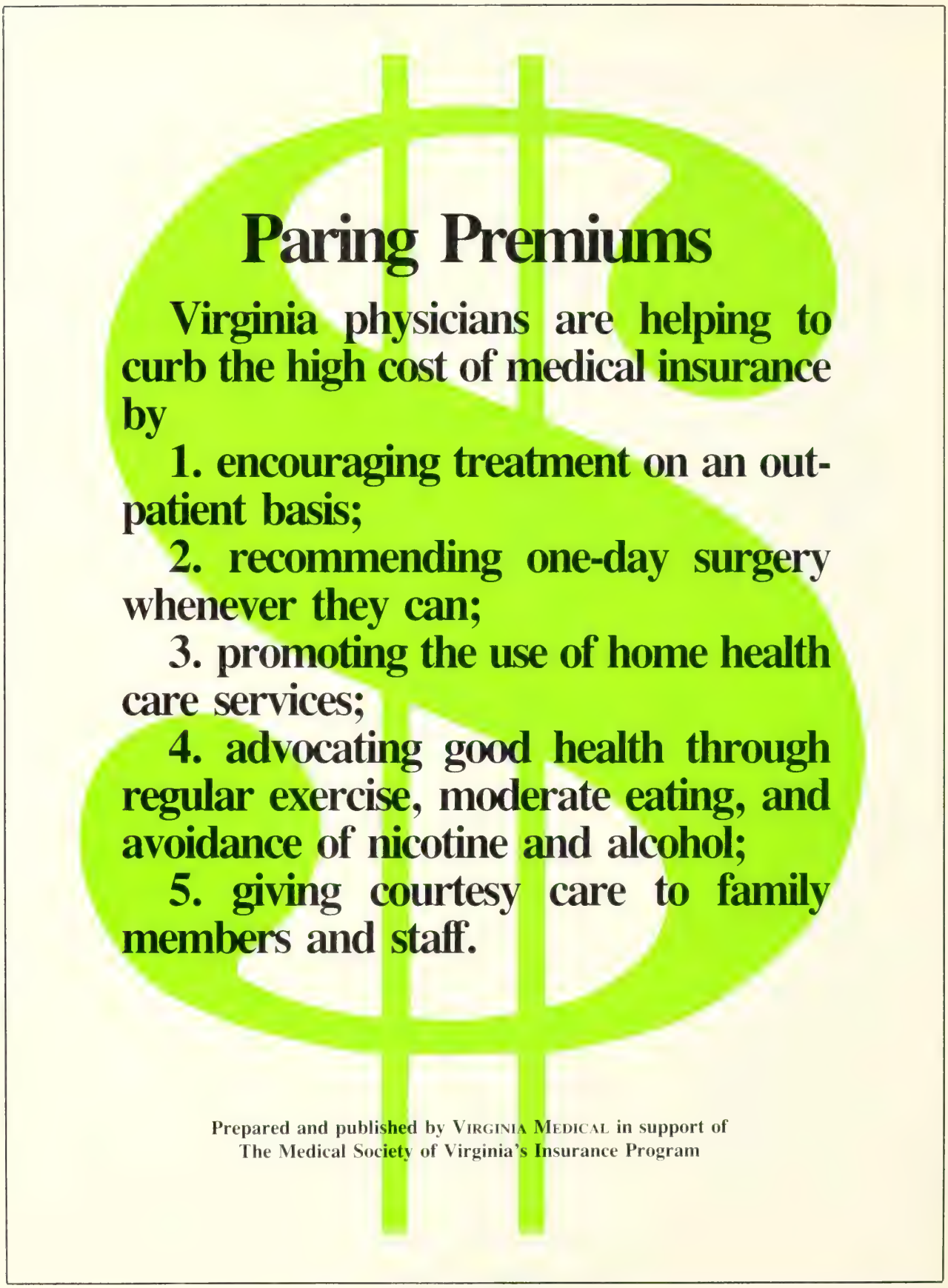
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EDITORIAL

Costs, Caps, and Contingencies

You can hear the old man now: "Things are not what they used to be". Well, they aren't—at least not in the health care field.

When I was a medical student there were no antibiotics, no corticosteroids, no intensive care units, no cardiac intensive care units, no open heart surgery, no microsurgery, no hip replacements, no fetal monitoring, no amniocentesis, no CAT scans—the list of major advances seems endless.

The number of medical schools has almost doubled and the number of graduates *has* doubled. Hospitals have proliferated and are now being operated with a cost-concious eye by a new breed, variously titled as president, executive vice-president, administrator, etc., all products of the newly created schools of hospital administration.

All of this denotes progress in the health care field, a great deal of progress, but there is a major fly in the ointment. You see headlines in your morning paper about it almost every day; the exploding costs of health care—almost 10% of the GNP. This frightening figure applies to the skyrocketing costs of the care of the hospitalized patient and is not necessarily applicable to the care of the ambulatory one (although there may be an occasional avaricious physician, too). The higher costs are the direct result of the many available advances in the field of medicine, with an assist from the inflation of the general economy.

What can be done to curb this spiralling cost? One suggestion is to ration medicine. As has been pointed out, this rationing effect has been accomplished in Great Britain by the simple expedient of providing too few physicians and too few hospitals. Obviously that approach is not practicable in the United States and it should be noted that such an approach would not be acceptable to everyone; some will necessarily suffer. But something must be done.[1]

Another major cause of the higher cost of medical care does not receive as much publicity and that is what prompted me to write this editorial. When I first practiced medicine—indeed, for almost all of my 30 years in that field—the physician-patient relationship was an amicable one; the physician was the patient's friend. At least it always appeared that way to me. It isn't that way now. In the present litigious society, the relationship is more likely to be an adversarial one, and the physician who is a victim of a lawsuit, or even the one who feels threatened with the possibility, will practice "defensive" medicine—utilizing unnecessary studies and procedures in order to protect himself/herself.

Although it will not necessarily serve to return the amicable physician-patient relationship, there is an answer to this problem, albeit one patently unpalatable to a segment of the legal profession. Since a cap, or ceiling, on the maximum punitive damage is already in effect in Virginia, the ultimate solution lies in the elimination of the contingency fee for the lawyer. This can be effected only by a change in the Code of Virginia. In order to accomplish such a goal, the medical profession must become more and more politically oriented, not only in promoting the election to public office of friends of medicine, but also in providing physicians themselves as active candidates for public office.

Edwin L. Kendig, Jr., MD

1. Kendig ELjr: Comment. Va Med 109:823, 1982

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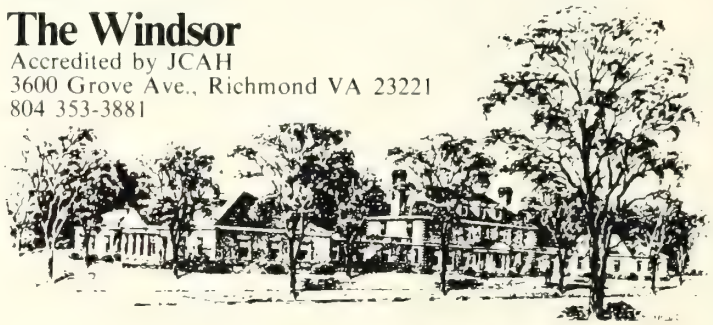

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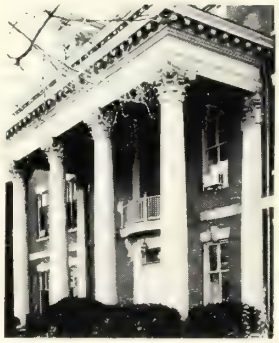

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VIRGINIA MEDICAL OBITUARY

Henry A. Kinser, MD

Dr. Henry A. Kinser, a past president of the Lee County Medical Society, died November 24, 1982, at Holston Valley Hospital and Medical Center, Kingsport, Tennessee. He was 60 years old.

A native of Dante, Virginia, Dr. Kinser was graduated from Lincoln Memorial University at Harrogate, Tennessee, and received his medical degree from the University of Tennessee Medical School, Memphis. He began his family practice in Pennington Gap in 1950, serving Lee County residents until his death. He was a veteran of World War II.

For 31 years a member of The Medical Society of Virginia, Dr. Kinser also belonged to the American Academy of Family Physicians.

Eldred S. Jones, MD

Dr. Eldred S. Jones, who over 55 years of general practice in Hampton had won great esteem in his community, died on January 13 at his Hampton home. He was 82 years old and had closed the office in his home four months earlier.

Born and reared in Hampton, Dr. Jones left his home city only once, to attend the University of Virginia and its School of Medicine, from which he was graduated in 1925. On the occasion of his retirement last year, a group of friends and relatives set up the Eldred S. Jones Symposium Fund to pay for day-long lectureships at Hampton General Hospital, where he hospitalized his patients.

Dr. Jones had belonged to The Medical Society of Virginia and the Hampton Medical Society since 1931.

A tribute to Dr. Jones was published in the editorial section of VIRGINIA MEDICAL's February issue[1], but he did not live to see it. It praised the high quality of his long practice, particularly his "unfaltering patience" and his "devotion to academic standards."

1. Miller JP: Hallmark of a long practice: quality. Va Med 110:128, 1983

Klaus W. Berblinger, MD

Dr. Klaus William Berblinger, who practiced psychiatry in Virginia for 16 years before moving to California in 1958, died on November 11, 1982, in San Francisco. He was 72 years old.

German born, Dr. Berblinger received his medical degree in 1934 from the Medizinische Fakultat der Ludwig Maximiliams Universität in Munich. He came to the United States to train at De Paul Hospital, Norfolk, then established a practice in Norfolk. In California, he was associated with the University of California Medical Center at San Francisco.

Dr. Berblinger became a member of The Medical Society of Virginia in 1940 and retained his membership throughout his California residence. He belonged also to the American Psychiatric Association, American College of Psychiatrists, and the Academy of Psychosomatic Medicine.

Grisha Jarandeh, MD

Dr. Grisha Jarandeh, Annandale, died on December 15, 1982, at the age of 42 years. A native of Iran, Dr. Jarandeh was graduated from the Faculty of Medicine of the University of Teheran and came to this country to intern at hospitals in the District of Columbia, then began practicing pediatrics in Alexandria. In addition to The Medical Society of Virginia, he belonged to the Alexandria Medical Society and the American Academy of Pediatrics.

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WHO'S WHO

WHEN HE IS INSTALLED as president of the American Academy of Neurology late this month in San Diego, **Dr. Nelson G. Richards** of Richmond will be the first private practitioner to lead the Academy. All the previous presidents have been professors, a natural sequence to the Academy's founding by academicians in the late 40s. The private practice of neurology has grown remarkably since then, and today the Academy counts 80% of its 7,250 members as clinical practitioners. Dr. Richards' selection as president reflects this shift in emphasis.

Born in New Jersey, Dr. Richards came to Virginia to attend the University of Virginia and went on to its School of Medicine, graduating in 1952. Training in internal medicine and neurology took him to the New York Hospital, Cornell University Medical Center, and he then spent a year at the Institutes of Health.

He began practicing at the Cleveland, Ohio, Clinic Foundation, where he established and was the first director of its laboratory of electroencephalography. When he moved to Roanoke in 1969, he was one of only eight practicing neurologists in Virginia; today they number 145.

For the past ten years Dr. Richards has practiced in Richmond and is currently a clinical professor of neurology at the Medical College of Virginia.

A believer in organized medicine, Dr. Richards was instrumental in the formation of the Virginia Neurological Society and has served as its president. He has also been president of the Society of Clinical Neurologists. He is a fellow of the American College of Physicians, and his other memberships include the American Society of Internal Medicine and the American EEG Society.

For the American Academy of Neurologists, Dr. Richards has performed numerous administrative chores, including service as secretary-treasurer, but his major contribution, his peers say, has been as chairman of the Practice Committee, which is the Academy's only standing committee. Dr. Richards convened this committee quarterly at airport hotels in what one colleague describes as "weekend lockups", with Dr. Richards' perserverance producing such major accomplishments as a brochure for patients explaining neurological disorders and care.

His peers predict that Nelson Richards will be a highly visible Academy president, and they expect his gentlemanly manner and communicative skills to make him a superior spokesman for the Academy.

These same attributes mark his hospitality when he and his wife, Sally, entertain, either at their Richmond home or their cottage on Taylor Creek. It is at the cottage that Nelson Richards relaxes and sets sail from the Rappahannock River Yacht Club, with Richmond an ocean away as the main and jib are raised on his 27-foot sloop. There may soon be more opportunities for these excursions when his son-in-law, John J. Hennessey IV, joins him in practice this summer.

The American Academy of Neurology has named its skipper and Nelson Richards is ready to set the course. Good sailing!

—MARY SELHORST

Cyane Lowden

Dr. Richards studying EEGs.

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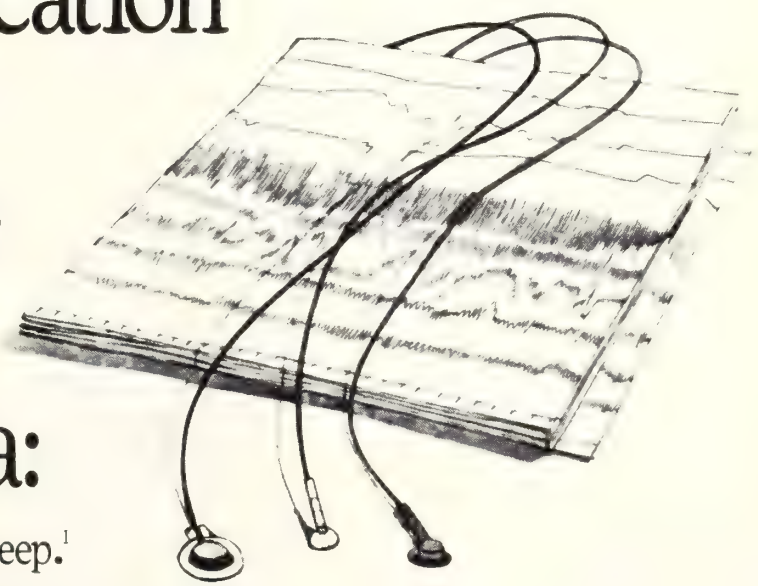
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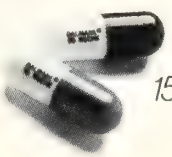

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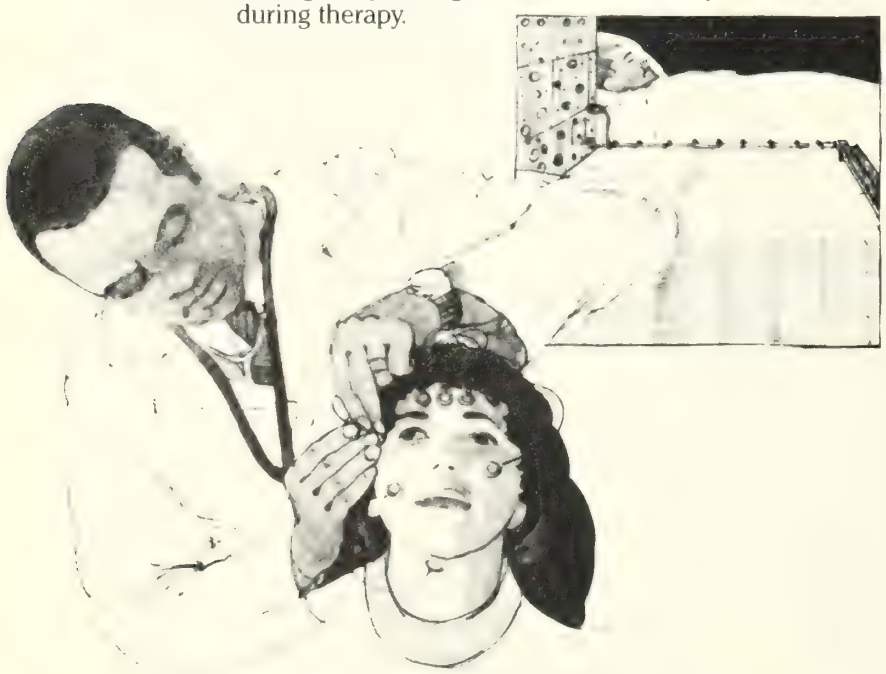

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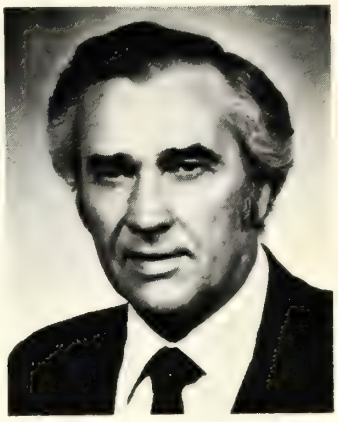

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May 1983, Volume 110, Number 5

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Virginia physicians invited to a PPO premier

"What we have now is not going to keep playing in Peoria. We've got to make a change."

So said Dr. Richardson Grinnan when he came to Medical Society of Virginia headquarters with an invitation to the Virginia premier of a preferred provider organization. It is being produced by Blue Cross Blue Shield of Virginia, its title is KeyCare, and it will start playing in the Richmond-Petersburg area on or about July 1.

Lanky and boyish-looking at 43, Dr. Grinnan became vice president of medical affairs for the Virginia Blues a year ago February. Before that he practiced as a pulmonary disease specialist at Richmond's McGuire Clinic. The Richmond medical community gives Dr. Grinnan a lot of respect. Says one leading Richmond physician, "We're lucky to have him there," i.e., at Blue Cross Blue Shield.

Cyane Lowden

Dr. Richardson Grinnan (standing) telling the PPO story. Seated at right, Dr. J. P. Anderson. Back to camera, Dr. Robert J. O'Donnell.

The audience for Dr. Grinnan's presentation was comprised of members of a Medical Society of Virginia committee called the Medicine/Business Coalition. Set up in 1981 when complaints from Big Business about the high cost of medical insurance were becoming strident in both Peoria and Richmond, the committee functions as liaison to Virginia's business community.

As chairman of the committee, Dr. Edward A. Zakaib, Richmond, introduced Dr. Grinnan. (For an editorial by Dr. Zakaib, see page 331.)

Around the conference table sat these committee members: Dr. J. P. Anderson, Waynesboro; Dr. G. Edward Calvert, Lynchburg; Dr. Russell D. Evett and Dr. Gervas S. Taylor, Jr., both of Norfolk; Dr. Robert J. O'Donnell, Falls Church; Dr. R. L. Sallade, Newport News; Dr. J. Latane Ware, Richmond; and Dr. Earl D. White II, Hampton.

They listened with unfaltering attention to Dr. Grinnan's 90-minute pitch. KeyCare is an economy model, was the message. Estimates forecast 10%-20% savings per subscriber group.

To generate these lower costs, the hospitals and physicians under contract as "preferred providers" will agree to tender services at contracted rates, and subscribers will kick in through copayment and deductible clauses.

Choosing his figures tentatively, Dr. Grinnan estimated 10,000-20,000 subscribers for starters. That's sufficient population for analysis of the plan and its experience. Of 12 hospitals in the Richmond area, at least five are expected to be placed under contract, he said. How many physicians will participate? Dr. Grinnan didn't know. Were there plans for other KeyCares in other Virginia settings? Yes, Virginia's Tidewater is the next target area, with early 1984 as the launch date.

Preferred provider organizations have evolved from health maintenance organizations, which were invented in California 20 years ago and became big box office across the country in the late 1970s, although physicians gave them decidedly mixed reviews.[1] In fact, KeyCare is Blue Cross Blue Shield's response to an HMO that opened for business in Richmond in 1982.[2] Its name: PruCare, after its owner and operator, the Prudential Insurance Company.

Like the HMO movement, the PPO movement appears to be aimed at the big consumer, i.e., business. And what do businessmen want these days in the medical insurance they buy in quantity? 1) A broad range of services for their broad range of employees. Both HMOs and PPO offer that. KeyCare will even offer dental coverage, if the subscriber already has the benefit under an existing Blues plan. 2) Low cost. Both HMOs and PPOs strive to make medical care less costly by contracting for the services of a specific group of doctors and hospitals at lower-than-usual fees and rates.

But then the two schemes diverge. Under PruCare, for instance, payment is not provided when subscribers use doctors or hospitals outside the contracted group, excepting referral by a participating physician or a medical emergency outside the contract area.

The KeyCare subscriber, however, can select a non-participating physician, or "non-par," but reimbursement is at 80% of the KeyCare schedule for the service rendered, and the money goes to the subscriber, from whom the non-par must collect.

Furthermore, HMOs are set up to avoid copayment and deductible devices, but PPOs can use them freely. Thus the KeyCare subscriber will be responsible for a $10 copayment for the outpatient services of a physician, and for outpatient diagnostic procedures, such as lab work or X-rays, the subscriber must pay 10% of the charges. To the participating physician goes the task of collecting these copayments.

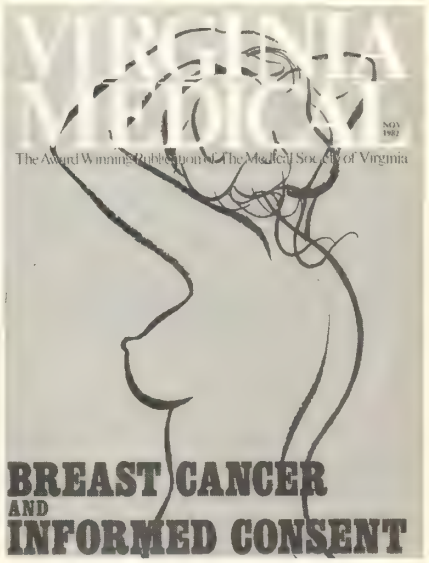
For hospitalization, the new PPO

Va Med keeps the prizes coming

From judges for the 1983 Sandoz Medical Journalism competition came a prize for VIRGINIA MEDICAL, a Special Award for the November 1982 issue on breast cancer and informed consent. "The sensitive subject was handled in an unorthodox manner, and the results were superb," said the Sandoz newsletter, *Medical Journalism*.

Craig D. Burrell, MD, Sandoz Pharmaceuticals vice president, sent notification of the award, which carries with it a $250 check. A Sandoz representative is to present the award at The Medical Society of Virginia's annual meeting in November at the Homestead.

In the eight years of the Sandoz contest's existence, VIRGINIA MEDICAL has twice won first prize, in 1977 and in 1981. The blue ribbon winner in the state medical journal category this year was the Journal of the Florida Medical Association.



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will specify a $200 deductible to be paid "up front", after which the plan pays the participating hospital's preset rate.

As for physicians' fees, including fees for inpatient surgery, KeyCare's projected schedule is at or near 85% of the current usual, customary, and reasonable reimbursement rate.

But to encourage surgery performed on an outpatient basis, or "ambulatory surgery," reimbursement is at 100% of the UCR.

For inpatient "global surgery," which is surgical lingo to embrace patient evaluation and preparation, the surgery itself, and followup care, a single reimbursement is to be developed. Reimbursement for history, physical exam, and days of care are not to be included unless performed by a general surgeon called in for consult.

For treatment in a hospital emergency room, there is to be a $30 copayment charge to the subscriber, but for treatment at one of the new, free-standing "emergicenters," the subscriber is to be assessed the $10 copayment that is standard for an office visit.

As is customary in an HMO, KeyCare's PPO providers are to undergo rigorous utilization review by Blue Cross Blue Shield, and each provider's contribution is to be analyzed to measure compatibility with the plan's goals.

"Will you still offer other plans?" Dr. Grinnan was asked after he had worked his way through the nuts and bolts of the PPO model.

"Oh sure," he answered jovially. "We'll be happy to sell you a Cadillac."

The appearance of the original preferred provider organization seems to have gone unheralded and unrecorded, but it is generally conceded to have occurred late in the 1970s in Denver. When word got out that it looked like a winner, PPOs began proliferating so swiftly that no one has solid data on how many of them there now are across the country.

In Denver, there are now four. When enabling legislation was passed last year in California, PPOs became a veritable contagion there, with at least 30 established to date and more in the planning stages.

Virginia's General Assembly passed similar enabling legislation a few months ago, giving the go-ahead to Blue Cross Blue Shield of Virginia's PPO ambitions. That legislation will be reported in full in VIRGINIA MEDICAL's coming June issue, when a review of the 1983 legislature's actions will be published.

The Virginia legislation does not have a portentous provision that goes into effect in California this coming July 1. It empowers California insurers to contract with so-called "exclusive provider organizations," or EPOs, which would operate similarly to PPOs but would not reimburse patients who choose providers outside the group.

California physicians are "frantic," Thomas C. Paton, president of Blue Shield of California, told an *American Medical News* reporter.[3] "They're feeling threatened that their style of living will be diminished. I can't say I blame them for being worried."

A California Blue Cross spokesman predicted to the same reporter that only the most efficient hospitals will survive once insurers begin contracting with hospitals on a flat-rate basis, and if physicians practice only at high-cost facilities, they'll "lose out" when the hospitals don't receive contracts.

The president of the San Francisco Medical Society, Dr. Mark Oscherwitz, said that doctors there are in a dilemma about whether to accept discounted fees in exchange for membership in a PPO.

"It's like sitting at the edge of the ballroom and being asked to dance. I'd want to look the prospects over very carefully before I accepted, but I'd be devastated if I weren't asked."

Dr. Charles M. Hair, president of the California Medical Association, was quoted as having an optimistic point of view. The large number of PPOs contracting for providers' services is "the epitome of free enterprise and the antithesis of national health insurance," he believes. "Given the choice, I'd rather have PPOs, assuming they're well organized and well run. That way we can still maintain the type of private health care delivery system people say they are happy with."[4]

Through his private practice, Dr. Hair belongs to a foundation for medical care and an independent practice association, and he is a member of Blue Shield.

"Eventually, I'll probably belong to a PPO as well," he said. "It's simply a matter of survival."

—ANN GRAY
and CARL L. SHIRES

1. Gray A: Virginia Medical's guide to HMO-watching. Va Med 107: 764-771, 1980
2. AG: McGuire Clinic, Prudential partner HMO in Virginia. Va Med 109:485, 1982
3. Staver S: New California PPOs producing tumult among physicians. Am Med News, February 25, 1983, pp 1, 12-15

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Brief Summary of prescribing information

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INDICATIONS AND USAGE: Ru-Tuss Tablets provide relief of the symptoms resulting from irritation of sinus, nasal and upper respiratory tract tissues.

CONTRAINDICATIONS: Hypersensitivity to antihistamines or sympathomimetics. Ru-Tuss Tablets are contraindicated in children under 12 years of age and in patients with glaucoma, bronchial asthma and women who are pregnant. Concomitant use of MAO inhibitors is contraindicated.

WARNINGS: Ru-Tuss Tablets may cause drowsiness. Patients should be warned of possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers.

PRECAUTIONS: Ru-Tuss Tablets contain belladonna alkaloids, and must be administered with care to those patients with urinary bladder neck obstruction. Caution should be exercised when Ru-Tuss Tablets are given to patients with hypertension, cardiac or peripheral vascular disease or hyperthyroidism. Patients should avoid driving a motor vehicle or operating dangerous machinery (See WARNINGS:).

OVERDOSAGE: Since the action of sustained release products may continue for as long as 12 hours, treatment of overdoses directed at reversing the effects of the drug and supporting the patient should be maintained for at least that length of time. Saline cathartics are useful for hastening evacuation of unreleased medication. In children and infants, antihistamine overdosage may produce convulsions and death.

ADVERSE REACTIONS: Hypersensitivity reactions such as rash, urticaria, leukopenia agranulocytosis, and thrombocytopenia may occur. Other adverse reactions to Ru-Tuss Tablets may be drowsiness, lassitude, giddiness, dryness of the mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, dizziness and insomnia. Large overdoses may cause tachypnea, delirium, fever, stupor, coma and respiratory failure.

DOSAGE AND ADMINISTRATION: Adults and children over 12 years of age, one tablet morning and evening. Not recommended for children under 12 years of age. Tablets are to be swallowed whole.

Federal law prohibits dispensing without prescription.

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NEW MEMBERS

Albemarle County Medical Society

Robert L. Fritz, MD, Family Practice, 1149 Rose Hill Dr., Charlottesville VA 22901

Worthington G. Schenk III, MD, General Surgery, Box 190, University of Virginia Medical Center, Charlottesville VA 22908

Augusta County Medical Society

Gerry D. Martin, MD, Ophthalmology, 428 S. Magnolia Ave., Waynesboro VA 22908

Fairfax County Medical Society

Jane W. Geltner, MD, Pediatrics/Pathology, 2501 Parker's Lane, Alexandria VA 22306

Kurt Lee Rhymers, MD, Obstetrics/Gynecology, 5606 Wood Thrust Court, Fairfax VA 22032

Fredericksburg Area Medical Society

Warren E. Parmelee, MD, Emergency Medicine, 2103 Cowan Blvd., Fredericksburg VA 22401

James River Medical Society

Devdass D. Karmarkar, MD, Family Practice, PO Box 8, Scottsville VA 24590

Medical Society of Northern Virginia

Mark A. Rhodes, MD, Orthopedic Surgery, 148 N. Main St., Woodstock VA 22664

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Richmond Academy of Medicine

Janice K. Brenneman, MD, Radiology, 1502 Chauncey Lane, Richmond VA 23233

James H. Cane, MD, Obstetrics/Gynecology, PO Box 26786, Richmond VA 23261

Richard G. Lewis, MD, Cardiology, 3500 Kensington Ave., Richmond VA 23221

Susan L. Williamson, MD, Radiology, 3500 Kensington Ave., Richmond VA 23221

Adrienne M. Young, MD, Internal Medicine, 956 Lisson Crescent, Richmond VA 23225

Roanoke Academy of Medicine

Louis J. Castern, MD, Emergency Medicine, 2913 Lockridge Rd., SW, Roanoke VA 24014

Southside Virginia Medical Society

Dorothy F. Cooney, MD, Radiology, Box 816, Farmville VA 23901

Randall H. Suslick, MD, Family Practice, 946 N. Main St., Chase City VA 23924

LETTERS

Malpractice data study yields comparison of Virginia experience

In a dispatch "From the Virginia Medical News Bureau,"[1] mention is made of the National Association of Insurance Commissioners study of closed claims in medical malpractice between the years 1975 and 1978.[2] From the "statistician's dream of charts, categories, percentages, averages, and other arithmetical indexes," a wealth of data can be gleaned with regard to where physicians in Virginia stood in those years in comparison with physicians across the nation.

The study covers all claims closed between July, 1975 and December, 1978. A closed claim is a claim that is paid or dismissed. Consequently, many claims covered in the study were originated before 1975 and many claims initiated during the years of the study were not closed during those years. In spite of these limitations, the study is quite complete. In these times of increasing concern and even outrage on the part of physicians about this issue, I believe it is important to attend to what data are available.

During the time of the study, 888 claims against physicians in Virginia were closed, of which 163 or 18.4% were paid claims. In the nation as a whole, 43,464 claims against physicians were closed, of which 16,429 or 38% were paid claims. Looking at the data from a different perspective, there were in Virginia 3.95 claims per 100 physicians per year and .72 paid claims per 100 physicians per year. In the nation as a whole, there were 3.83 claims per 100 physicians per year and 1.45 paid claims per 100 physicians per year. Therefore, with regard to malpractice claims against physicians in the years 1975 to 1978, physicians in Virginia were about as likely to be involved in a closed claim as were physicians across the country. However, Virginia physicians were about 50% as likely to be involved in a *paid* closed claim. The average amount paid out for a paid closed claim was $23,762 in Virginia and $25,161 across the nation.

With regard to large paid claims, those of $100,000 or more, ten such claims were settled in Virginia during the time of the study. There were therefore .04 large claims per 100 physicians per year in Virginia. Nationally, there were 1,066 large claims settled, or .09 large claims per 100 physicians per year.

Another onerous aspect of medical malpractice cases is the long time that elapses between the incident that gives rise to the case and the ultimate disposition of the case. During the time of the study, the average elapsed time in Virginia from the time of the incident until the filing of the claim was reported to the malpractice insurer was 11 months for a paid claim and 12 months for a claim that was closed without payment. In the nation as a whole, the elapsed time from the time of the incident to the time of the reported claim was 17 months for a paid claim and 16 months for an unpaid claim.

In Virginia, the average elapsed time from the time of the incident to the time of final disposition was 27 months for paid claims and 25 months for unpaid claims. Nationally, 41 months was the average time from the time of the incident to the time of disposition for paid claims, while 32 months was the average for unpaid claims. Virginia physicians were thus subjected to a somewhat shorter period of time during which a claim was active.

No less than physicians across the country, physicians in Virginia continue to experience medical malpractice suits as a difficult and upsetting aspect of delivering medical care. However, data from the National Association of Insurance Commissioners Study demonstrate that Virginia physicians were at some advantage relative to physicians across the country in regard to 1) the probability of being involved in a paid claim and 2) a relatively shorter elapsed time between the time of the incident that gave rise to the claim and the time that the case was ultimately closed.

Paul A. Mansheim, MD
Crawford Parkway at Fort Lane
Portsmouth VA 23704

1. From the Va Med News Bureau: Malpractice bills from plaintiffs' bar to resurface. Va Med 109:3-5, 1982
2. Malpractice Claims: Medical Malpractice Closed Claims, 1975-1978, Volume 2, No. 2. Brookfield, Wisconsin, National Association of Insurance Commissioners, 1980.

Annals of anecdotal medicine: a problem in passing urine

The news of the death in Montreal on October 16, 1982, of Hans Selye, MD, brought to mind his visit to Richmond in the 1950s to lecture to students and faculty of the Medical College of Virginia. Dr. Selye was then head of the Institute of Experimental Medicine and Surgery, where his research had made him an international figure in medicine.

He talked on his favorite subject, stress, and during the course of his remarks he told a story which I have recalled with amusement over the years. It later appeared in his book, *From Dreams to Discovery*, published in 1964 by McGraw Hill Book Company, as enclosed.

Kinloch Nelson, MD
Office of Continuing Medical Education
The Medical Society of Virginia
4205 Dover Road, Richmond VA 23221

Editors' Note: The story is reprinted below by permission of the Hans Selye Foundation of the International Institute of Stress in Montreal.

THE HANDICAP OF INTERNATIONAL BORDERS. It happened during the reign of His Most Gracious Majesty King George VI, that I was desperately looking for the urine of patients suffering from periarteritis nodosa. I suspected that this disease might be due to a derangement in the adaptive activity of the adrenals and associated with an increased corticoid excretion. Now, I wanted to verify this hypothesis, but at that time the disease was not very well known and probably not often diagnosed. In any event, we could not find a single case either in Montreal or in the vicinity; eventually I located two cases in Burlington, Vermont, just across the border. Because of the war, we were on a strict austerity program, and it was considered unpatriotic to import valuable materials from the United States, but, pushed by curiosity, I decided to proceed anyway. Suitable arrangements were made for shipment of the urine in sealed containers by plane so that it might arrive fresh. One of my associates was to meet the plane and I was waiting in my lab ready to extract the specimen before any decomposition could set in.

During this vigil I received a desperate phone call from my assistant at the airport informing me that the Royal Customs men could not pass urine. I wanted to know why and learned that the item was simply not listed in His Majesty's Customs Book, either as duty-free or as dutiable; hence, it could not pass. Irritated by this silly red tape, I asked our dean to write an official letter to the highest customs authorities of the land, explaining that, in basic research, we sometimes need items which might impress people as odd, but we need them, just the same. The customs officials of a cultured nation should know more about the needs of research.

This strongly worded letter, coming from a dean of McGill University, worked. Only a few days later we received an answer informing us that, of course, the federal authorities had foreseen such cases; it was only the inexperienced customs official at the airport who didn't know where to look for this item in the book. It was listed there clearly as duty-free, since it was manifestly comprised by the category described as "used personal articles."

Now this was fine, but by then I knew the shipment had decomposed anyway, so I did not collect. During the following days, we received more and more urgent telephone calls from the airport, begging us to remove the merchandise which apparently had become somewhat offensive. I didn't budge, because by that time I had lost interest. Finally, the postman brought a printed statement; evidently such cases had also been foreseen, or there would not have been such a form to fit the occasion. Only the number of my shipment was inscribed in handwriting and the printed text read as follows: "Unless within five (5) days after receipt of this notice you collect the merchandise mentioned above, the shipment will be opened and contents sold at public auction."

I must admit I failed to follow the case further and, to this day, I do not know who the happy winner was. But I do believe that my tragic experience clearly indicates the need for better public understanding of the nature and special requirements of basic research.

At Westbrook Hospital, we recognize the importance of family communication. Our specialized treatment programs for adolescents, adults and the elderly, focus on the needs of the individual and the family. A wide range of therapeutic services help families identify issues, share expectations, and talk about tomorrow.

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Anxious patients improve in just a few days

And what is more reassuring to an excessively anxious patient than medication that promptly starts to relieve his discomforting symptoms? Valium® (diazepam/Roche) begins working within 30 to 90 minutes. Patients continue to improve in just a few days, and relief continues throughout the course of treatment.

There are other important benefits with Valium as well—along with its broad clinical range, Valium has an efficacy/safety profile that few, if any, drugs can match. This record has been achieved with extensive clinical experience, undoubtedly including yours. And, as you must have observed, side effects more serious than drowsiness, fatigue or ataxia rarely occur. Nevertheless, as with any CNS-acting agent, patients should be cautioned about driving, operating hazardous machinery or ingesting alcohol or other CNS-depressant drugs while taking Valium.

Yet another benefit Valium affords is flexibility. Available in 2-mg, 5-mg and 10-mg scored tablets, Valium enables you to titrate dosage to individual patient needs. For the geriatric patient, a starting dosage of 2 to 2½ mg once or twice a day is recommended. And, for patients who forget or skip medication, you can prescribe Valrelease™ (diazepam/Roche) 15-mg slow-release capsules, knowing that Valrelease will assure all the benefits of Valium 5 mg *t.i.d.* with the convenience of once-a-day dosage.

Discontinuation of Valium (or Valrelease) is typically as smooth as its start in short-term therapy. However, Valium and Valrelease should be discontinued gradually after more extended treatment. As you diminish dosage, the built-in tapering action of Valium and Valrelease will help avoid rapidly recurring anxiety symptoms and symptoms of withdrawal, and will help ease the patient's transition to independent coping when therapeutic goals have been achieved.

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For a summary of product information, please turn the page.

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Valium® (diazepam/Roche) Ⓝ Tablets
Valrelease™ (diazepam/Roche) Ⓝ slow-release Capsules
Injectable Valium® (diazepam/Roche) Ⓝ

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, impending or acute delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: relief of skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome. *Oral forms* may be used adjunctively in convulsive disorders, but not as sole therapy. *Injectable form* may also be used adjunctively in: status epilepticus; severe recurrent seizures; tetanus; anxiety, tension or acute stress reactions prior to endoscopic/surgical procedures; cardioversion.

The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence

> *Usage in Pregnancy:* **Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins, i.e., dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia.

In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Dosage: Individualize for maximum beneficial effect.

ORAL *Adults:* Anxiety disorders, relief of symptoms of anxiety—Valium (diazepam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose.

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.)

For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children *administer I.V. slowly*; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful.

In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

How Supplied:

ORAL Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules—15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

INJECTABLE Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative.

ROCHE®

Physicians who recognize the importance of referral for patients with psychiatric, drug or alcohol problems will recognize the importance of this page.

Portsmouth Psychiatric Center and Norfolk Psychiatric Center are modern facilities providing services that are recognized as being the highest quality available in the country. Our treatment teams develop separate, individualized programs that provide adults, young adults, and children 5 through 18 with the environment and care necessary for treatment and recovery. And our Education Department holds a certificate to operate from the Board of Education, allowing adolescents, young adolescents and children to continue their education while hospitalized.

If you are seeing a patient with any of these problems, your referral to us will help them receive the necessary treatment to put them on the road to recovery and back to a normal life. Admissions and referral services are available to any referring physician, patient, or family member, 24 hours a day, 7 days a week. The cost for treatment at both hospitals is covered by most major insurance companies, including Blue Cross and Champus. Medicare is accepted at Norfolk Psychiatric Center. Our business office is available to answer specific questions on coverage.

To arrange admissions, or for more information, call us at the numbers below. We're Norfolk and Portsmouth Psychiatric Centers, and we can help.

Psychiatric, drug and alcohol problems are difficult, but there is help available.

Norfolk Psychiatric Center

Granby Street and Kingsley Lane, Norfolk, Virginia 23505
Telephone (804) 489-1072

Portsmouth Psychiatric Center

Fort Lane and Crawford Parkway, Portsmouth, Virginia 23704
Telephone (804) 393-0061

Accreditation: *Joint Commission on Accreditation of Hospitals.* **Membership:** *American Hospital Association, Federation of American Hospitals, National Association of Private Psychiatric Hospitals, Virginia Hospital Association, Tidewater Hospital Council.* **Licensure:** *Commonwealth of Virginia Department of Mental Health and Mental Retardation.*

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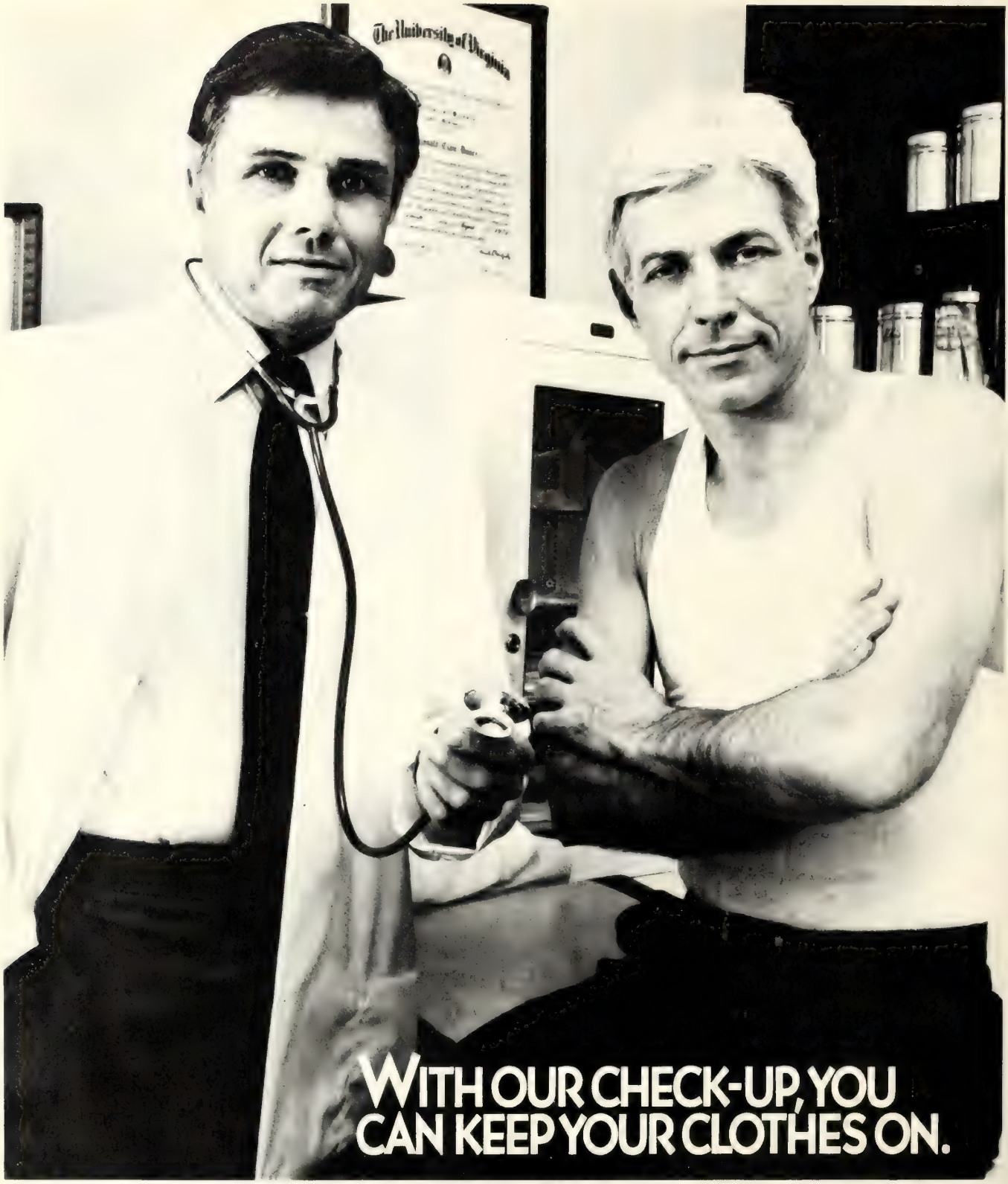

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See important information on next page

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tablets

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Effective analgesic/anxiolytic alliance

Proven superior to aspirin alone in controlled clinical trials

(BRIEF SUMMARY)

DESCRIPTION. Each tablet contains 200 mg meprobamate and 325 mg aspirin

INDICATIONS: Adjunct in short-term treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease. Clinical trials demonstrated in these situations relief of pain is somewhat greater than with aspirin alone. Effectiveness in long-term use, i.e. over 4 months, has not been assessed by systematic clinical studies. Physicians should periodically reassess usefulness of drug for individual patients.

CONTRAINDICATIONS. ASPIRIN: Allergic or idiosyncratic reactions to aspirin or related compounds. MEPROBAMATE: Acute intermittent porphyria, allergic or idiosyncratic reactions to meprobamate or related compounds, e.g. carisoprodol, mebutamate, or carbromal.

WARNINGS. ASPIRIN: Use salicylates with extreme caution in patients with peptic ulcer, asthma, coagulation abnormalities, hypoprothrombinemia, vitamin K deficiency, or those on anticoagulants. In rare instances, aspirin in persons allergic to salicylates may result in life threatening allergic episodes. MEPROBAMATE: DRUG DEPENDENCE: Physical and psychological dependence, and abuse have occurred. Chronic intoxication from prolonged ingestion of, usually, greater than recommended doses is manifested by ataxia, slurred speech, and vertigo. Therefore, carefully supervise dose and amounts prescribed and avoid prolonged use, especially in alcoholics and others with known propensity for taking excessive quantities of drugs. Sudden withdrawal after prolonged and excessive use may precipitate recurrence of preexisting symptoms, e.g. anxiety, anorexia, or insomnia, or withdrawal reactions, e.g., vomiting, ataxia, tremors, muscle twitching, confusional states, hallucinosis, and, rarely, convulsive seizures. Such seizures are more likely in persons with CNS damage or preexistent or latent convulsive disorders. Onset of withdrawal symptoms occurs usually within 12 to 48 hours after discontinuation, symptoms usually cease within next 12-to-48-hour period. When excessive dosage has continued for weeks or months, reduce dosage gradually over 1 to 2 weeks rather than stop abruptly. Alternatively, a short-acting barbiturate may be substituted, then gradually withdrawn.

POTENTIALLY HAZARDOUS TASKS: Warn patients meprobamate may impair mental or physical abilities required for potentially hazardous tasks, e.g., driving or operating machinery.

ADDITIVE EFFECTS: Since CNS-suppressant effects of meprobamate and alcohol or meprobamate and other psychotropic drugs may be additive, exercise caution with patients taking more than one of these agents simultaneously.

USAGE IN PREGNANCY AND LACTATION. An increased risk of congenital malformations associated with minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during first trimester of pregnancy, has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at time of institution of therapy should be considered. Advise patients if they become pregnant during therapy or intend to become pregnant to communicate with their physicians about desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breastfeeding patients, consider the drug's higher concentrations in breast milk as compared to maternal plasma levels.

USAGE IN CHILDREN: Keep preparations with aspirin out of reach of children. Equagesic®-M is not recommended for patients 12 years of age and under.

PRECAUTIONS: ASPIRIN: Salicylates antagonize uricosuric activity of probenecid and sulfinpyrazone. Salicylates are reported to enhance hypoglycemic effect of sulfonylurea antidiabetics.

MEPROBAMATE: Use lowest effective dose, particularly in elderly and/or debilitated, to preclude over-sedation. Meprobamate is metabolized in the liver and excreted by the kidney, to avoid excess accumulation exercise caution in its use in patients with compromised liver or kidney function. Meprobamate occasionally may precipitate seizures in epileptic patients. It should be prescribed cautiously and in small quantities to patients with suicidal tendencies.

ADVERSE REACTIONS. ASPIRIN: May cause epigastric discomfort, nausea, and vomiting. Hypersensitivity reactions, including urticaria, angioneurotic edema, purpura, asthma, and anaphylaxis may rarely occur. Patients receiving large doses of salicylates may develop tinnitus.

MEPROBAMATE: CNS: Drowsiness, ataxia, dizziness, slurred speech, headache, vertigo, weakness, paresthesias, impairment of visual accommodation, euphoria, overstimulation, paradoxical excitement, fast EEG activity.

GI: Nausea, vomiting, diarrhea.

CARDIOVASCULAR: Palpitation, tachycardia, various forms of arrhythmia, transient ECG changes, syncope, hypotensive crisis.

ALLERGIC OR IDIOSYNCRATIC: Milder reactions are characterized by itchy, urticarial, or erythematous maculopapular rash, generalized or confined to the groin. Other reactions include leukopenia, acute nonthrombocytopenic purpura, petechiae, ecchymoses, eosinophilia, peripheral edema, adenopathy, fever, fixed drug eruption with cross-reaction to carisoprodol, and cross-sensitivity between meprobamate, mebutamate and meprobamate-carbromal. Rare, more severe hypersensitivity reactions include hyperpyrexia, chills, angioneurotic edema, bronchospasm, oliguria, and anuria. Also, anaphylaxis, exfoliative dermatitis, stomatitis, and proctitis. Stevens-Johnson syndrome and bullous dermatitis have occurred.

HEMATOLOGIC (SEE ALSO "ALLERGIC OR IDIOSYNCRATIC"): Agranulocytosis, aplastic anemia have been reported, although no causal relationship has been established, and thrombocytopenic purpura.

OTHER: Exacerbation of porphyric symptoms.

DOSAGE AND ADMINISTRATION: Usual dose is one or two tablets, 3 to 4 times daily as needed for relief of pain when tension or anxiety is present. Not recommended for patients 12 years of age and under.

OVERDOSAGE: Treatment is essentially symptomatic and supportive. Any drug remaining in the stomach should be removed. Induction of vomiting or gastric lavage may be indicated. Activated charcoal may reduce absorption of both aspirin and meprobamate. Aspirin overdosage produces usual symptoms and signs of salicylate intoxication. Observation and treatment should include management of hyperthermia, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions. Suicidal attempts with meprobamate have resulted in drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse. Some suicidal attempts have been fatal. The following data, reported in the literature and from other sources, are not expected to correlate with each case (considering factors such as individual susceptibility and length of time from ingestion to treatment), but represent usual ranges reported. Acute simple overdose (meprobamate alone): Death has been reported with ingestion of as little as 12 gram meprobamate and survival with as much as 40 gram.

BLOOD LEVELS

0.5-2.0 mg percent represents usual blood-level range after therapeutic doses. The level may occasionally be as high as 3.0 mg percent.

3-10 mg percent usually corresponds to findings of mild-to-moderate symptoms of overdosage, such as stupor or light coma.

10-20 mg percent usually corresponds to deeper coma, requiring more intensive treatment. Some fatalities occur.

At levels greater than 20 mg percent, more fatalities than survivals can be expected.

Acute combined overdose (meprobamate with other psychotropic drugs or alcohol): Since effects can be additive history of ingestion of a low dose of meprobamate plus any of these compounds (or of a relatively low blood or tissue level) cannot be used as a prognostic indicator.

In cases of excessive doses, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Any drug remaining in stomach should be removed and symptomatic treatment given. Should respiration or blood pressure become compromised, respiratory assistance, CNS stimulants and pressor agents should be administered cautiously as indicated. Diuresis osmotic (mannitol) diuresis, peritoneal dialysis, and hemodialysis have been used successfully in removing both aspirin and meprobamate. Alkalinization of the urine increases excretion of salicylates. Careful monitoring of urinary output is necessary, and caution should be taken to avoid overhydration.

Relapse and death, after initial recovery, have been attributed to incomplete gastric emptying and delayed absorption.

HOW SUPPLIED: Bottles of 50 scored tablets.

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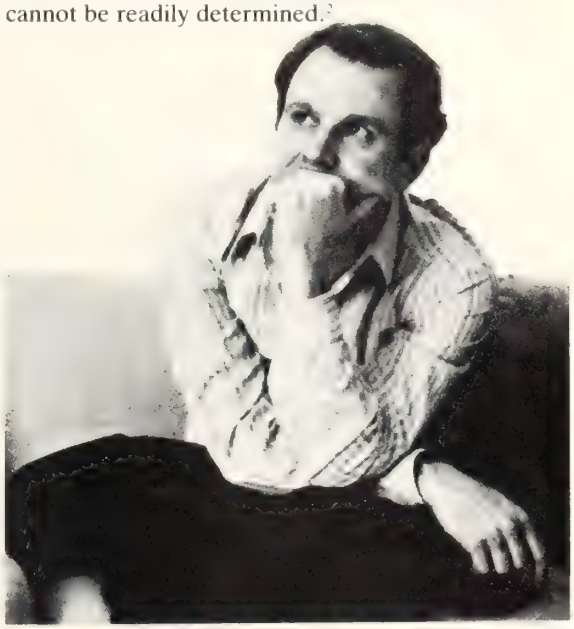

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1. Dixon RE: *Ann Intern Med* 89(Part 2):749-753, Nov 1978. 2. Shils ME, Randall HT: Diet and nutrition in the care of the surgical patient, chap. 36, in *Modern Nutrition in Health and Disease*, edited by Goodhart RS, Shils ME; Philadelphia, Lea & Febiger, 1980, p. 1114

Please see summary of product information on reverse page.

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THE MULTIVITAMIN/MINERAL FORMULATION

Before prescribing, please consult complete product information, a summary of which follows:

Each Berocca® Plus tablet contains 5000 IU vitamin A (as vitamin A acetate), 30 IU vitamin E (as *dl*-alpha tocopheryl acetate), 500 mg vitamin C (ascorbic acid), 20 mg vitamin B_1 (as thiamine mononitrate), 20 mg vitamin B_2 (riboflavin), 100 mg niacin (as niacinamide), 25 mg vitamin B_6 (as pyridoxine HCl), 0.15 mg biotin, 25 mg pantothenic acid (as calcium pantothenate), 0.8 mg folic acid, 50 mcg vitamin B_{12} (cyanocobalamin), 27 mg iron (as ferrous fumarate), 0.1 mg chromium (as chromium nitrate), 50 mg magnesium (as magnesium oxide), 5 mg manganese (as manganese dioxide), 3 mg copper (as cupric oxide), 22.5 mg zinc (as zinc oxide)

INDICATIONS: Prophylactic or therapeutic nutritional supplementation in physiologically stressful conditions, including conditions causing depletion, or reduced absorption or bioavailability of essential vitamins and minerals; certain conditions resulting from severe B-vitamin or ascorbic acid deficiency, or conditions resulting in increased needs for essential vitamins and minerals

CONTRAINDICATIONS: Hypersensitivity to any component

WARNINGS: Not for pernicious anemia or other megaloblastic anemias where vitamin B_{12} is deficient. Neurologic involvement may develop or progress, despite temporary remission of anemia, in patients with vitamin B_{12} deficiency who receive supplemental folic acid and who are inadequately treated with B_{12}.

PRECAUTIONS: *General.* Certain conditions may require additional nutritional supplementation. During pregnancy, supplementation with vitamin D and calcium may be required. Not intended for treatment of severe specific deficiencies. *Information for the Patient.* Toxic reactions have been reported with injudicious use of certain vitamins and minerals. Urge patients to follow specific dosage instructions. Keep out of reach of children. *Drug and Treatment Interactions.* As little as 5 mg pyridoxine daily can decrease the efficacy of levodopa in the treatment of parkinsonism. Not recommended for patients undergoing such therapy.

ADVERSE REACTIONS: Adverse reactions have been reported with specific vitamins and minerals, but generally at levels substantially higher than those in Berocca Plus. However, allergic and idiosyncratic reactions are possible at lower levels. Iron, even at the usual recommended levels, has been associated with gastrointestinal intolerance in some patients.

DOSAGE AND ADMINISTRATION: Usual adult dosage: one tablet daily. Not recommended for children. Available on prescription only.

HOW SUPPLIED: Golden yellow, capsule-shaped tablets — bottles of 100.

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MEETINGS

June 2-3
Treating the Difficult Alcoholic Patient: Avoiding Malpractice (University of Maryland), *Baltimore*. Michael Wessely, Education Coordinator, 10 S. Pine St., Baltimore MD 21201, (301) 528–3956.

June 6-10
7th Annual Myocutaneous Flap Dissection Workshop (Plastic Surgery Specialists, Inc.), *Norfolk*. Vicky Martin, 400 W. Brambleton Ave., Norfolk VA 23510, (804) 623-1090.

June 8-12
Rehabilitation of the Brain-Injured Adult (Medical College of Virginia), *Richmond*. 22 credit hrs. Fee, $225. Sheri Rosner, Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 780-0494.

June 9-11
Advances in Pediatric Ophthalmology and Strabismus (Eastern Virginia Medical School), *Virginia Beach*. Earl Crouch, MD, EVMS, PO Box 1980, Norfolk VA 23510.

June 11
Ambulatory Surgical Units, Their Organization and Function (Medical College of Virginia), *Richmond*. Surinder K. Kallar, MD, Box 695, MCV Station, Richmond VA 23298, (804) 786-9160.

June 11-12
Practical Dermatology for the Non-Dermatologist (University of North Carolina), *Williamsburg*. 7 hrs. Cat. I. Fee, $75. Virginia Moore, UNC School of Medicine, Dept. of Dermatology, Chapel Hill NC 27514.

June 13-17
Advanced Practitian in Ultrasound (Johns Hopkins University), *Baltimore*. Fee, $400. Julia C. Burbridge, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

June 13-17
9th Annual Family Medicine Review Course (University of Maryland), *Baltimore*. Terry S. Young, Office of CME, 10 S. Pine St., Baltimore MD 21201.

June 19-24
Eastern Shore Medical Symposium (University of Delaware/Medical Society of Delaware), *Rehoboth Beach, Delaware*. Fee, $395. Sylvia Brocka, Division of CME, University of Delaware, 2800 Pennsylvania Ave., Wilmington DE 19806, (302) 738-8151.

June 23-30
Sports and Tennis Medicine Symposium (Georgetown University/Virginia Sports Medicine and Rehabilitation Institute), *Wimbledon, England.* Cat. I. Robert P. Nirschl, MD, 3801 N. Fairfax Dr., Arlington VA 22203, (703) 525-8600.

June 24-26
Internal Medicine Symposium (Eastern Virginia Medical School), *Virginia Beach.* James P. Baker, MD, EVMS, PO Box 1980, Norfolk VA 23510.

June 30-July 2
Dermatology Days (University of Maryland), *Ocean City, Maryland,* 11 credit hrs. Fee, $190. Terry S. Young, Office of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

July 15-17
Ambulatory Electrocardiography (International Medical Education Corporation), *Virginia Beach.* 13 credit hrs. Fee, $260. IMEC, 64 Inverness Dr., E, Englewood CO 80112, toll free, (800) 525-8651.

July 22-24
Advances in Cardiology (Medical College of Virginia), *Hot Springs.* 12 credit hrs. Fee, $230. Randy Casey, Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

9th ANNUAL
FAMILY MEDICINE REVIEW COURSE
June 13-17, 1983
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For further information contact:

Program of Continuing Education
University of Maryland
School of Medicine
10 South Pine Street
Baltimore, Maryland 21201
(301) 528-3956

July 22-24
Annual Golf Classic Symposium: Selected Topics for the Primary Care Physician (Eastern Virginia Medical School), *Williamsburg.* Robert L. Cassidy, MD, EVMS, PO Box 1980, Norfolk VA 23501.

July 28-30
Pediatrics at the Beach: Diagnostic and Management Problems (Medical College of Virginia), *Virginia Beach.* Fee, $235. Randy Casey, Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

July 29-31
Internal Medicine Summer Seminar (Eastern Virginia Medical School), *Virginia Beach.* James P. Baker, MD, EVMS, PO Box 1980, Norfolk VA 23510.

SERIAL LEARNING PROGRAMS

Four one-week courses in medical radiation safety will be offered in June by the University of Virginia's Radiological Physics Division:

June 6-11 Basic Radiation Safety
June 13-18 Radiation Safety in Diagnostic Radiology
June 20-25 Radiation Safety in Radiotherapy
June 27-July 2 Radiation Safety in Nuclear Medicine

Courses may be taken independently or in combination, at $300 for each course. For further information: Suresh K. Agarwal, PhD, Box 375, University of Virginia Medical Center, Charlottesville VA 22908.

Colby College in Waterville, Maine, has presented continuing medical education courses since 1945, building a national reputation for courses of quality in a pleasurable summer milieu. Physicians and their families are accommodated on campus. The courses offered in 1983 are listed below; all are accredited for Category 1. For further information, write to Robert H. Kany, Colby College, Waterville ME 04901, or call (207) 873-1131.

July 5-9	Dermatology
July 11-15	Pediatrics
July 12-15	Surgical Techniques
July 14-15, 18-19	Computer Course
July 19-23	Infectious Disease
July 25-29	Emergency Medicine
July 25-29	Otolaryngology
August 1-4	Anesthesiology
August 1-5	Pulmonary Disease
August 7-11	Ophthalmology
August 8-12	Obstetricis/Gynecology
August 12-14	Forensic Medicine
August 14-19	Nuclear Medicine
September 12-16	Marine Medicine

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The Adult Chemical Dependency Treatment Program deals with individual patient needs in a non-psychiatric approach that involves intense hospital care and patient/family education and counseling. Emphasis is on communication and non-chemical coping skills without the use of psychotropic drugs.

The Adolescent Chemical Dependency Treatment Program places special emphasis on the adolescent and the family, with therapy designed to help restructure the family unit and aid in the recovery of all. The full services of Peninsula Academy allow for continued education while the patient undergoes treatment.

The family program for Adolescent and Adult Chemical Dependency focuses on helping concerned individuals adjust to living with another's alcohol or drug problem. The program's non-psychiatric approach emphasizes family education to the problem, enhanced by individual and group therapy with an aim toward positive development of the whole self.

As with all Peninsula Psychiatric Hospital's programs, there are no eligibility restrictions with regard to race, religion, sex, residency or disability. For details on any of our Chemical Dependency Treatment Programs, telephone **Hampton (804) 722-2504, Norfolk (804) 622-5577, Richmond (804) 796-2016.**

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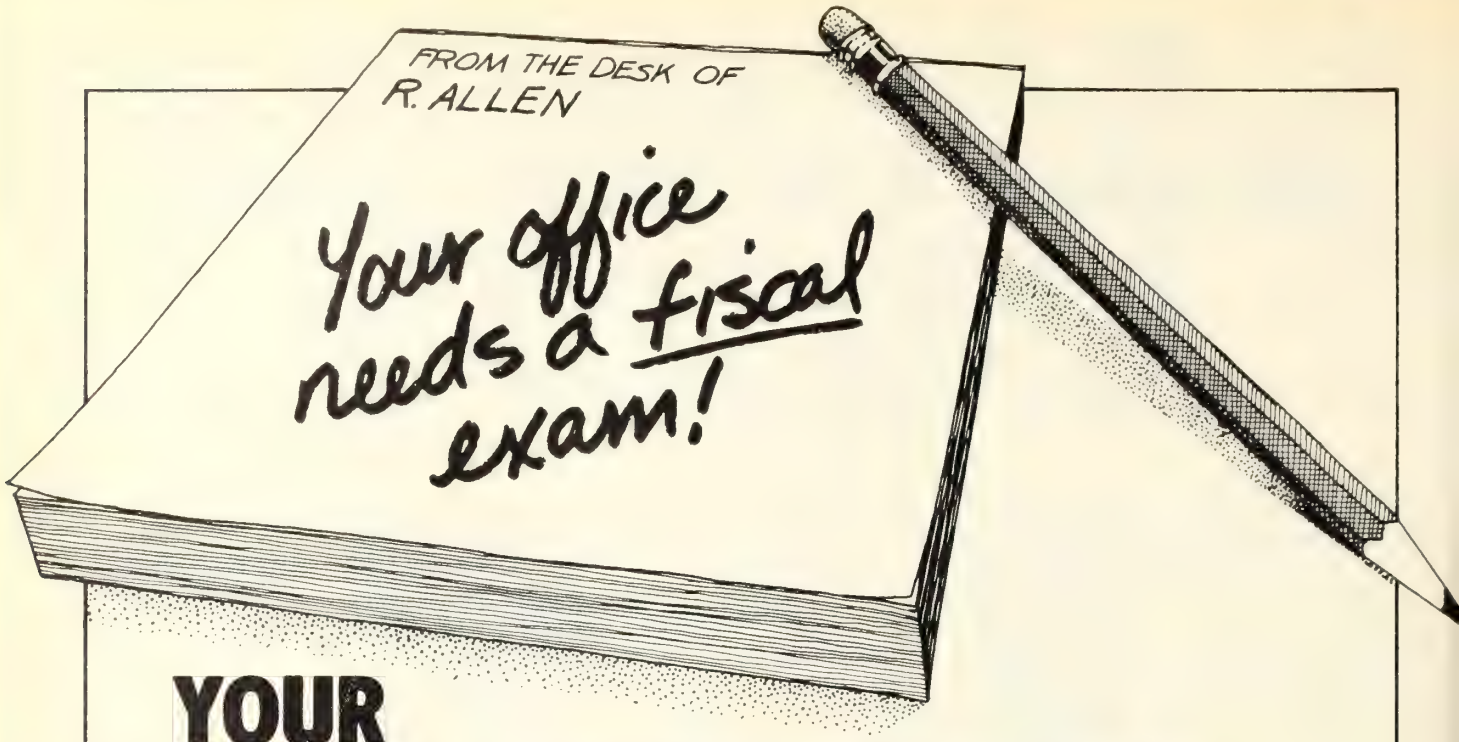

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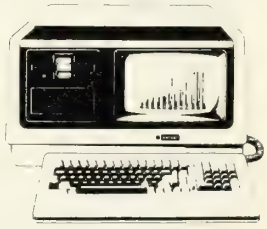

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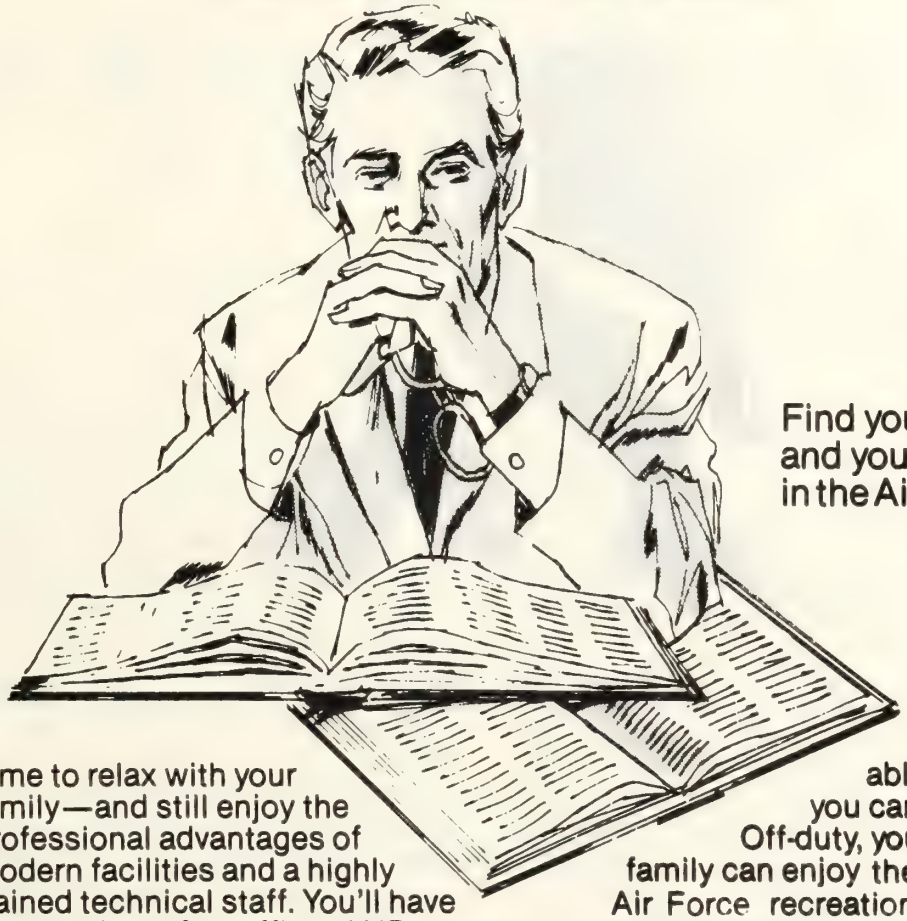

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MEDICAL
MAY 1983

Diagnosing Gastrointestinal Bleeding with Labeled Red Cells

John S. Mustol, MD, Robert E. Berry, MD, Marshall A. Wakat, MD, and Bruce D. Baird, MD, *Roanoke, Virginia*

The red blood cells of 62 patients suspected of gastrointestinal bleeding were labeled with technetium-99m and then tracked by scintigraphy. Active bleeding was confirmed in 20 and their lesions identified and localized. The authors recommend the technique for patients with intermittent or slow-to-moderate lower GI hemorrhage, as in hematochezia; as a screening test for angiography; and for patients who refuse other studies or are allergic to contrast media.

THE development of angiography and endoscopy has greatly improved the detection and localization of gastrointestinal bleeding. In the past few years, a radionuclide scan using technetium-99m labeled red cells has come into use for this purpose. With this study we present our experience with this scan and make suggestions as to its use in the diagnosis of gastrointestinal bleeding.

Materials and Methods

From September 1979 to March 1982, scintillation scans were performed on 62 consecutive patients suspected of upper or lower GI hemorrhage at Roanoke Memorial Hospitals (ages 11 months to 93 years, average age 63.5 years, 32 males and 30 females). Twenty-four patients were referred with hematochezia, 20 with melena, eight with occult blood in their stools, six with bright red blood in their emesis, and four with coffee ground material in their emesis. No patients were in shock or had unstable vital signs. Using a technique developed by Pavel and Zimmer[1], a patient was given an intravenous injection of stannous pyrophosphate (0.2 mg/kg) while in his hospital room or on arrival in the nuclear medicine department. Thirty minutes later a second IV injection of 15-20 mCi of technetium-99m pertechnetate was given. This technique produces a radioactive label on the patient's red cells which lasts about 24 hours and is the same technique used for gated heart studies in cardiac patients. A large field of view scintillation camera was used for imaging.

Immediately after injection a flow study scinti-

From Roanoke Memorial Hospitals, Roanoke VA 24033. Address correspondence to Dr. Berry.

Submitted 6-28-82.

gram of the patient's abdomen was obtained, followed by static images at 5- to 15-minute intervals for one hour. If no abnormal collection of tracer was identified, further imaging was obtained at various intervals up to 24 hours post-injection. Bleeding was identified by the appearance of extravasated labeled red cells in the gastrointestinal tract. Patients were observed throughout their hospital stay and on followup. In general, no attempt was made by the authors to influence the clinical management of the patient. As controls, 13 patients undergoing gated heart studies with no clinical evidence of gastrointestinal bleeding were scanned at four and 24 hours post-injection.

The use of this red-cell labeling technique for localization of GI bleeding was first reported by Winzelburg et al in 1979[2].

Results

Of the 13 control patients, the presence of small amounts of radioactivity in the colon was revealed at four hours in one patient and 24 hours in eight patients. The intensity of this activity was never greater than that of the iliac vessels, which were used as a reference for background activity level. This low level of colon activity is believed due to incomplete binding of pertechnetate to red cells or the slow loss of pertechnetate from the surface of red cells with secretion into the GI tract and accumulation in the colon. The radiologist must be aware of this occurrence in order to avoid overreading late images leading to false-positives. Newer techniques of in vitro labeling provide more efficient binding of pertechnetate to the red cells, helping to avoid this problem[3]. We are now using in vitro labeling at our institution.

The results of the scans of the 62 bleeding patients were compared to clinical data including serial hematocrits, transfusion requirements, nasogastric aspirates, stools, barium contrast studies, angiography, endoscopy, surgical findings and autopsy findings. Twenty-nine of the 62 patients were thought to be actively bleeding at the time of the scan on the basis of falling hematocrits, continued bloody stools, continued transfusion requirements, or active bleeding demonstrated by another study such as angiography or endoscopy.

The technetium-99m labeled red cell scan correctly identified the bleeding sites in 20 of the 62 patients (32%) suspected of GI bleeding as confirmed by the above clinical data. Of the 29 patients thought to be actively bleeding, 20 bleeding lesions (69%) were correctly diagnosed and successfully localized, as follows: duodenal ulcer, five; colon diverticulum, four; carcinoma of the colon, marginal ulcer, and Meckel's diverticulum, two each; hemorrhagic gastritis, gastric ulcer, angiodysplasia of the cecum, aortoduodenal fistula, and esophageal varices, one each.

The scans of eight patients (13%) were interpreted as false-positive when the scan showed evidence of bleeding and other clinical data indicated the patient was not bleeding, or the scan showed evidence of bleeding at a site in the GI tract other than that identified by other clinical studies.

Fourteen patients (23%) had scans interpreted as true-negative. These scintigrams showed no evidence of bleeding, and this was confirmed by the clinical courses of the patients and other clinical studies. These scans were classified as true-negative even if a potential bleeding site was identified, such as a non-bleeding ulcer on endoscopy, as long as there was no clinical evidence of active bleeding during the time the scan was being done.

There were no false-negative scans.

Twenty patients (32%) were deemed to have had insufficient additional clinical data collected to confirm or refute the scans' findings. These were classified as "no correlation".

Lesions missed by the scan either because they were not bleeding or due to a false positive scintigram were these: duodenal ulcer, three; colon polyp, two; angiodysplasia of the cecum, hemorrhagic gastritis, gastric ulcer, and gastric carcinoma, one each.

After retrospectively assessing the clinical courses of all patients excluding those classified as "no correlation", the scan was deemed helpful in the diagnosis of 23 patients, neither helpful nor harmful in 13, and misleading in six. Angiography was performed in 11 patients in this series, correctly identifying the bleeding site in four. Esophagogastroduodenoscopy was done in 24 patients, correctly demonstrating the bleeding site or probable bleeding site in 15. Colonoscopy was done in 15 patients showing the active or potential bleeding site in five patients.

Figure 1 is the 24-hour image of a 78-year-old man with hematochezia showing evidence of bleeding in the cecum. Earlier images had shown a suggestion of activity in the cecum. Angiography and colonscopy were negative. Because of continued bleeding, a right colectomy was performed and a vascular ectasia was identified in the cecum on pathological examination. The patient has had no further bleeding. Figure 2 is the image 10 minutes after injection of an 11-month-old boy with hematochezia. It

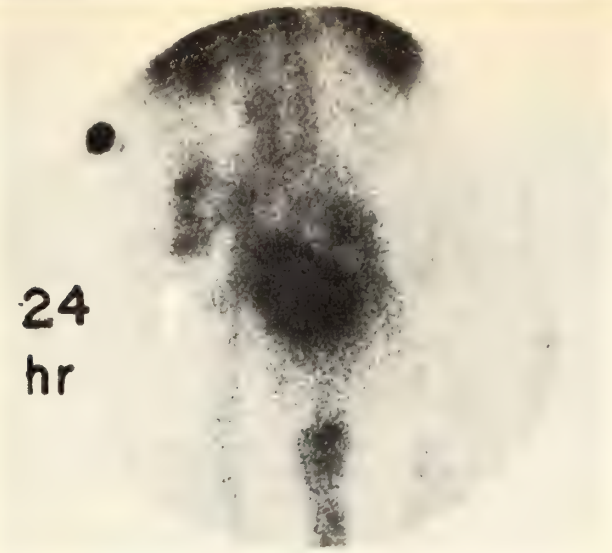

Fig. 1. Lower abdominal image 24 hours post-injection of 78-year-old man with hematochezia. Arrow indicates intense activity in cecum.

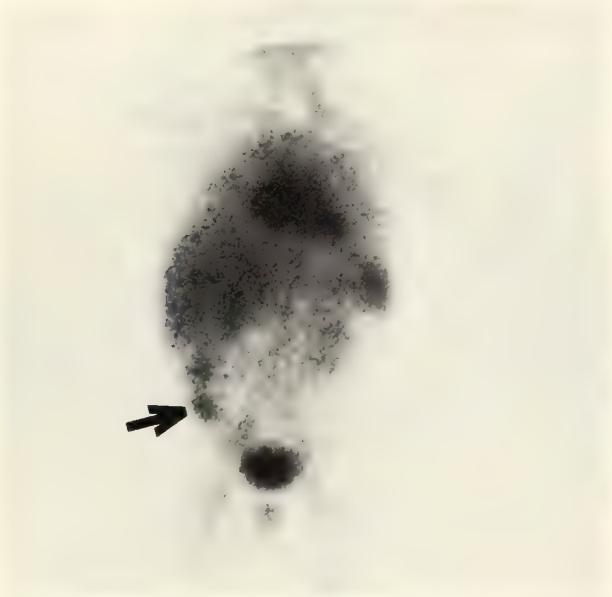

Fig. 2. Ten minutes postinjection image of 11-month-old child with hematochezia. Arrow shows activity in cecum indicating bleeding in distal small bowel or cecum.

Fig. 3. Thirty-minute image of 76-year-old man with hematochezia. Arrow indicates bleeding from left colon with labeled blood coursing up and down left colon.

shows evidence of bleeding in the region of the cecum, and at surgery a Meckel's diverticulum was removed. Figure 3 is the scintigram 30 minutes post-injection of a 76-year-old man with hematochezia. It demonstrates bleeding from the left colon. The patient had known diverticuli of the right and left colon. After a left colectomy, he had no further bleeding. In this instance, the scan permitted localization of diverticular bleeding to one side of the colon, so that a total colectomy could be avoided.

Of eight patients presenting with occult blood in their stools, the bleeding site was localized in one. Two of 20 patients presenting with melena had their bleeding sites correctly localized, and 14 of 24 patients presenting with hematochezia were localized using the scan. Of 17 patients whose scans were done on a stat or emergency basis, bleeding sites were correctly localized in 13. Of 45 patients whose scans were done electively, bleeding sites were localized in only seven.

Discussion

Recent reports have presented data indicating a high degree of sensitivity for the labeled red cell scan in the diagnosis of gastrointestinal hemorrhage[2-5]. The purpose of the present report is to present data on an unselected consecutive series of patients and to offer some guidelines to the physician as to the clinical usefulness of the scan.

In our series, the scan proved highly sensitive. Surprisingly, we had no false-negative scans. Our data indicate the scan lacks specificity, with eight false-positive scintigrams. This is probably due to inefficient in vivo labeling or loss of label from the red cells as noted above in the control patients. This should improve with use of in vitro labeling techniques[2]. Three of the eight false-positive scans showed bleeding in the GI tract distal to the actual site of bleeding as determined by other clinical studies. Extravasated red cells may be propelled rapidly through the GI tract to the cecum or rectum producing activity in these areas on delayed images. The skill and experience of the radiologist is very important in interpreting delayed images.

On the basis of our experience, we feel the scan is useful in intermittent or slow to moderate lower GI hemorrhage, that is, patients of any age with hematochezia. As noted by Winzelburg et al[2], it is often difficult to coordinate a bleeding episode with angiography. The radionuclide scan provides a 24-hour period during which the blood pool is labeled, and scintigrams may demonstrate a site of bleeding any time during that 24 hours. The scan may localize

venous GI bleeding as well, which is difficult to demonstrate using angiography.

The scan may also be useful as a screening test for angigoraphy since it is more sensitive but less specific than angiography. In a patient whose rate of bleeding is slow and there is doubt as to whether or not angiography will be fruitful, a red cell scan may be performed. If scintigrams were positive in the first five to ten minutes, angiography could be expected to show positive findings, or may not be required.

The labeled red cell scan may also be useful in children and patients who refuse other studies or who are allergic to contrast media. The procedure is simple, minimally invasive, fast, and requires equipment available in most nuclear medicine departments. Radiation dosage is about that of an adult PA and lateral chest X-ray.

Although ten patients in our series had upper GI lesions localized by the radionuclide scan, we do not feel it is a first-line test in the diagnosis of upper GI hemorrhage. At the present time, endoscopy seems to be the best diagnostic tool for upper GI hemorrhage since it can demonstrate not only the location of the bleeding, but also the nature of the source, such as an ulcer, a carcinoma, or varices. If endoscopy is fruitless, then the scan may be useful as a screening test prior to angiography as noted above.

A total of 28 patients with either melena or occult blood in their stools had scans, and only three were positive. Therefore, we do not feel the labeled red cell scan is useful, at least as a first-line study, in patients with this clinical presentation. Barium contrast studies and endoscopy would seem more useful in this setting.

We also recommend that the study be done as an emergency procedure when the patient first presents in the emergency room or shows signs of active GI bleeding. Thirteen of 17 (76%) emergency scans were positive in our series, whereas only seven of 45 (16%) elective scans were positive. The patient must be actively bleeding for the scan to be successful. If the scan is done electively the next morning, the patient will often stop bleeding with a night of bed rest and empiric therapy, and the scan will be negative.

Bowel preps for barium enema or colonoscopy may flush extravasated red cells out of the GI tract, leading to false-negative scans. Although this did not happen in our series, we recommend that patients not receive enemas or purgatives while scanning is in progress.

Lastly, we emphasize that the skill and experience of the radiologist performing and interpreting the scans is of great importance. We have found that the degree of usefulness of the scan to the clinician depends on the ability of the radiologist, clinical information in hand, to say what the images show.

Summary

Results are presented of a consecutive series of 62 patients with gastrointestinal hemorrhage utilizing in vivo technetium-99m labeled red blood cells to detect and localize the bleeding. Bleeding sites were correctly localized in 20 of 29 actively bleeding patients (69%). On the basis of this experience the labeled red cell scan is felt to be useful in patients of any age with slow to moderate lower GI hemorrhage; as a screening procedure for angiography; in children and patients who refuse other studies or who are allergic to contrast media. We do not believe the scan is a first-line test in the diagnosis of upper GI hemorrhage or in patients presenting with melena or occult blood in their stools. We recommend that the scan be done on an emergency basis and that purgatives and enemas be avoided during scanning. Finally, the skill and experience of the radiologist is very important in interpreting the scans and making them clinically useful.

We wish to thank Miss Kathy Kiger and Mr. Ed Hamilton for their help in preparing this paper.

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Sclerotherapy of Bleeding Esophageal Varices

Paul Monroe, MD, *Richmond, Virginia*

Reviewing a 14-month experience with 38 sclerotherapy procedures on 17 patients with bleeding esophageal varices, the author documents an 87% effectiveness rate and risks he finds acceptable. It is an old technique, the author notes, revived recently when the flexible endoscope became available.

Hemorrhage from esophageal varices is the most serious complication of cirrhosis of the liver, which is the fourth most common cause of death in urban centers in the United States[1] and is associated with a 25%-50% hospital mortality.[2,3] Vasopressin therapy and balloon tamponade, the standard initial therapy for bleeding esophageal varices, may temporarily control hemorrhage but do not prevent rebleeding, nor do these forms of therapy improve hospital mortality.[4,5] Emergency portacaval shunts have a mortality as high as 50%, even in experienced hands.[6] Elective portacaval shunts prevent bleeding from esophageal varices but contribute to the development of hepatic failure and encephalopathy and therefore have not improved survival in controlled studies. The distal splenorenal shunt has been used since it selectively decompresses esophageal varices and thus reduces the incidence of hepatic failure and encephalopathy postoperatively.[7] Unfortunately, it is difficult to perform, requires an experienced surgeon, and may result in the late development of encephalopathy.[8]

Several centers have revived an old technique, endoscopic sclerotherapy of varices, for both the treatment of acute variceal bleeding and prevention of recurrent bleeding from esophageal varices. In recent years use of a flexible endoscope has made this procedure relatively simple and much safer than in initial studies of bleeding esophageal varices, when the rigid endoscope was used. Several studies have demonstrated that acute variceal bleeding can be controlled by sclerotherapy of esophageal varices in 87%-92% of patients, even in those not responding to balloon tamponade.[9,10] More importantly, a recent prospective randomized trial of chronic therapy in 107 patients showed a threefold decrease in bleeding and improved survival in the group receiving sclerotherapy compared to

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controls.[11] This study suggests that sclerotherapy may be a significant advance in the treatment and prevention of hemorrhage from esophageal varices.

This paper reviews my experience with 38 sclerotherapy procedures on 17 patients performed over 14 months, January 1981 through March 1982, at the Medical College of Virginia and McGuire Veterans Administration Medical Center. I have found sclerotherapy to be effective in 87% of patients treated so far and to have an acceptable complication rate.

Results

Sclerotherapy was performed with an Olympus GIF-Q or 1T endoscope. A 4-mm, 25-gauge needle on the end of a polyethylene catheter was used for injection. Intravariceal injections of 1-2 cc of 5% sodium morrhuate with 0-5 units of topical thrombin per cc were made. Varices were initially injected at the gastroesophageal junction, and further injections were made more proximally; usually 10-15 injections were made at each session. All patients had a complete blood count, chest radiography and electrocardiogram performed before and after the procedure. Patients were usually sclerosed twice in the first week and at 3-4 week intervals thereafter until all varices were obliterated. Chronic followup endoscopy was performed at 6-12 month intervals, and repeat sclerotherapy performed as necessary. All patients were treated with antacids and elevation of the head of their bed for two weeks after sclerotherapy in an attempt to reduce esophageal ulcers.

Eleven sclerotherapy procedures were performed acutely either at the diagnostic endoscopy or less than six hours after vasopressin or tamponade therapy was initiated. In nine of eleven patients the combined treatment was successful in stopping bleeding. Seven sclerotherapy treatments were performed within 48 hours of the bleeding (one patient had two procedures because of rebleeding from varices). Twenty other sclerotherapy treatments were performed chronically in patients to assure obliteration of the varices.

Of the 17 patients who underwent sclerotherapy, two had unsuccessful treatments; one died of bleeding, one had a portacaval shunt. Twelve had no further bleeding. Three had one minor episode of bleeding before obliteration of varices. I have found that two to five sclerotherapy sessions over 1-2 months are necessary to obliterate all varices. Bleeding prior to obliteration, which occurs in about 20% of patients, may be from varices or from esophageal ulcers induced by sclerotherapy; fortunately, it is often minor and easily controlled. Thus about 70% of patients have had no rebleeding after sclerotherapy (mean followup of five months), and a further 18% have had only minimal bleeding prior to obliteration of the varices and no bleeding since then.

Five patients (30%) have died: two from liver failure, one from pulmonary edema, one from metastatic tumor and one from uncontrolled bleeding. In each of the three patients who had autopsies, pathologists found organized clot in the esophageal varices. Two other patients I am following have had angiography after sclerotherapy which revealed very small or absent esophageal varices, although gastric varices persisted. The autopsy and angiography findings confirm my clinical impression that sclerotherapy is successful in thrombosing esophageal varices.

The most serious complication of endoscopic sclerotherapy was bleeding caused by the sclerotherapy, which stopped in two hours and required transfusion of two units of blood. Shortly after sclerotherapy, a second patient developed adult respiratory distress syndrome, which may have been caused by aspiration or sclerotherapy.

We are performing further studies to determine if sodium morrhuate and other sclerosing agents are toxic to the pulmonary vasculature since there is evidence that they circulate systemically after injection into the varix. Sodium morrhuate is a particularly effective sclerosing agent but does cause local toxicity, which may lead to deep ulceration of the esophagus. A few case reports have described systemic reactions to injected morrhuate.

Other agents have been utilized, such as ethanolamine oleate (not available in the United States but widely used in Great Britain), sodium tetradecyl sulfate, cephalothin sodium (Keflin®), and 50% dextrose. Other complications of sclerotherapy noted in the literature include deep esophageal ulceration leading to mediastinal perforation in 3%-5% of patients and esophageal strictures in 5%-10% of patients who undergo chronic sclerotherapy.[10-15] Less serious complications in my series include fever greater than 100° F (24%), pleural effusion (12%), esophageal ulcer (8%), and chest pain (58%). Most of these minor complications resolve in one or two days, although ulcers may persist for weeks.

Discussion

Endoscopic sclerotherapy was first performed in 1939 but was not used extensively until 1970. Large series reported from Europe involve over 900 patients treated in the last decade.[10] Several small

series have recently been reported in the United States.[12-14] The effectiveness and complication rate in these uncontrolled series is comparable to my own: about 84%-93% success rate for control of bleeding, about 20% minor rebleeding prior to obliteration of varices, and a 5%-10% risk of serious complications.[10-14]

Unfortunately only one large, well-performed, controlled series has studied the efficacy of this treatment. This prospective controlled study of 107 patients randomized to sclerotherapy or medical management found a threefold decrease in rebleeding and significantly improved survival at one year.[11] Once obliteration of varices was complete, 65% of patients reexamined at one year did not develop recurrence of esophageal varices. Those varices which recurred were reinjected. These results, although very encouraging, need to be confirmed at other centers.

A poorly controlled series from South Africa also found sclerotherapy to be of value in preventing rebleeding.[15] Recently, a preliminary report[16] of 12 patients randomized to sclerotherapy or esophageal transection found no significant difference in survival (33% vs 0% respectively). Although the number of patients in this study was very small and only critically ill patients were entered, this study points out the need for further investigation of the efficacy of sclerotherapy.

There is great variability in the methods used for performing sclerotherapy and further investigation is necessary to determine the optimal technique. Some authors prefer intravariceal injection,[11-16] while others believe perivariceal or submucosal injections are best.[10] The flexible endoscope gained favor with most sclerotherapists because it reduces complications and does not require general anesthesia. Some also utilize balloon tamponade devices proximal[16] or distal[15] to the injection site, while others do not. Finally, many agents have been used to induce thrombosis of the esophageal varices, but objective studies have not yet compared these agents. We are presently performing a series of studies in order to determine the best sclerosing agent for this purpose.

Unfortunately, few studies have been performed comparing these technical aspects of sclerotherapy. Differences in the effectiveness of sclerotherapy and the complication rate between investigators may be the result of technique and experience.

Sclerotherapy appears to have acceptable risks and to be useful for the treatment of actively bleeding esophageal varices that do not respond to usual medical management. Perhaps the most promising use of sclerotherapy may be the chronic treatment of patients who have bled from esophageal varices in order to prevent rebleeding. MacDougall's controlled study,[11] as well as uncontrolled experience such as the data presented here, suggest that rebleeding may be dramatically reduced in these patients.

Randomized comparisons of sclerotherapy with portacaval shunt, distal splenorenal shunt and esophageal transection are being performed at several centers and hopefully will help determine which patients should be selected for sclerotherapy. Further long-term, well-designed, controlled studies of the technique and its efficacy are necessary to define the role of sclerotherapy in the routine treatment of bleeding esophageal varices and to determine the safest and most effective sclerosing agent and method for successful sclerotherapy.

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BRIEF SUMMARY
PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug Interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request

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"I can do things that I couldn't do for 3 yrs. including joining the human race again."

Quotes from an unsolicited letter received by Pfizer from an angina patient.
While this patient's experience is representative of many unsolicited comments received, not all patients will respond to Procardia nor will they all respond to th[illegible]

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"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA[*] *as soon as it became available. The change in my condition is remarkable."*

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks,[1] taking fewer nitroglycerin tablets,[2] doing more, and being more productive once again

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%)

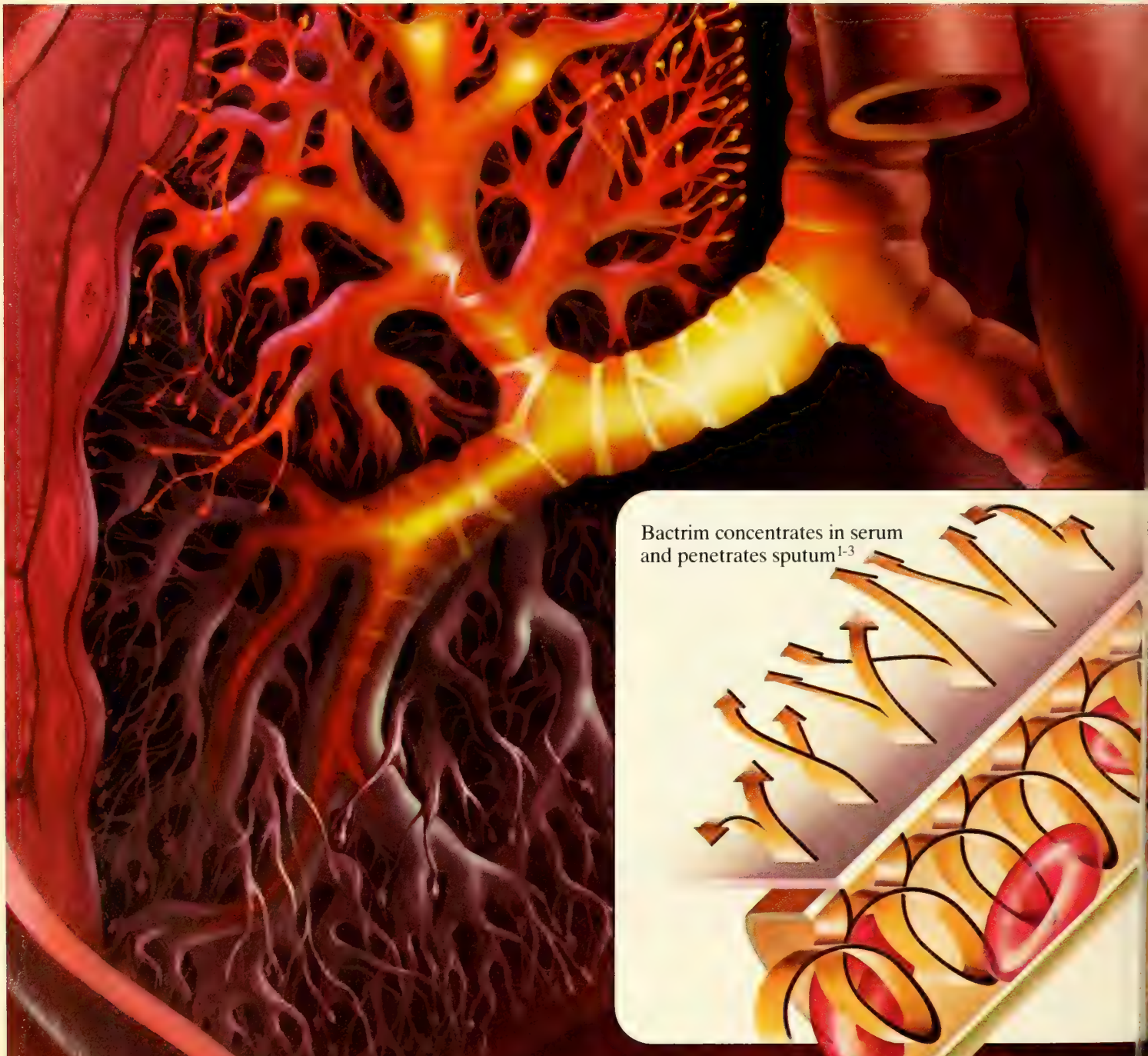
for the varied faces of angina

PROCARDIA®
(NIFEDIPINE) *Capsules 10 mg*

*Procardia is indicated for the management of.
1) Confirmed vasospastic angina.
2) Angina where the clinical presentation suggests a possible vasospastic component.
3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

Please see PROCARDIA *brief summary on adjoining page*

Bactrim™ attacks the
(trimethoprim and sulfamethoxazole/Roche)
in acute exacerbations



Bactrim concentrates in serum and penetrates sputum[1-3]

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ROCHE

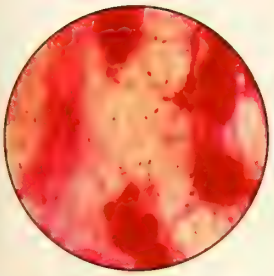
major pathogens of chronic bronchitis*

Bactrim clears sputum of susceptible bacteria

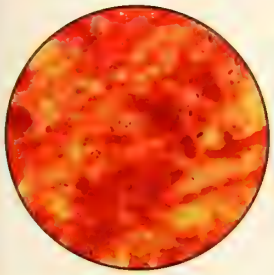
In sputum cultures from patients with acute exacerbations of chronic bronchitis, *H. influenzae* and *S. pneumoniae* are isolated more often than any other pathogens.[4,5] One study of transtracheal aspirates from 76 patients with acute exacerbations found that 80% of the isolates were of these two pathogens.[5]

Bactrim is effective *in vitro* against most strains of both *S. pneumoniae* and *H. influenzae*—even ampicillin-resistant strains. And in acute exacerbations of chronic bronchitis involving these two pathogens, sputum cultures taken seven days after a two-week course of therapy showed that Bactrim eradicated these bacteria in 91% (50 of 55) of the patients treated.[6]

attacks *H. influenzae*—even ampicillin-resistant strains



attacks *S. pneumoniae*



Bactrim reduces coughing and sputum production

In three double-blind comparisons with ampicillin *q.i.d.*, Bactrim DS proved equally effective on all clinical parameters.[7-9] Bactrim reduced the frequency and severity of coughing, reduced the amount of sputum produced and cleared the sputum of purulence.

Bactrim has the added advantages of *b.i.d.* dosage convenience and a lower incidence of diarrhea than with ampicillin, and it is useful in patients allergic to penicillins.

Bactrim also proved more effective than tetracyclines in 10 clinical trials involving nearly 700 patients.[10] Overall clinical condition of the patients, changes in sputum purulence, reduction in sputum volume and microbiological clearance of pathogens—all improved more with Bactrim therapy than with tetracyclines. G.I. side effects occurred in only 7% of patients treated with Bactrim compared with 12% of tetracycline-treated patients. (See Adverse Reactions in summary of product information on next page.)

Bactrim is contraindicated in pregnancy at term and nursing mothers, infants under two months of age, documented megaloblastic anemia due to folate deficiency and hypersensitivity.

Bactrim DS. For acute exacerbations of chronic bronchitis in adults* when it offers an advantage over single-agent antibacterials.

References: 1. Hughes DTD, Bye A, Hodder P: *Adv Antimicrob Antineoplastic Chemother 1/2*:1105-1106, 1971. **2.** Jordan GW *et al: Can Med Assoc J 112*:91S-95S, Jun 14, 1975. **3.** Beck H, Pechere JC: *Prog Antimicrob Anticancer Chemother 1*:663-667, 1969. **4.** Quintiliani R: Microbiological and therapeutic considerations in exacerbations of chronic bronchitis, in *Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts;* Princeton Junction, NJ, Communications Media for Education, Inc., 1980, pp. 9-12. **5.** Schreiner A *et al: Infection 6*(2):54-56, 1978. **6.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **7.** Chodosh S: Treatment of acute exacerbations of chronic bronchitis: results of a double-blind crossover clinical trial, in *Chronic Bronchitis and Its Acute Exacerbations: Current Diagnostic and Therapeutic Concepts. Op. cit.*, pp. 15-16. **8.** Chervinsky P: Double-blind clinical comparisons between trimethoprim-sulfamethoxazole (Bactrim™) and ampicillin in the treatment of bronchitic exacerbations. *Ibid.*, pp. 17-18. **9.** Dulfano MJ: Trimethoprim-sulfamethoxazole vs. ampicillin in the treatment of exacerbations of chronic bronchitis. *Ibid.*, pp. 19-20. **10.** Medici TC: Trimethoprim-sulfamethoxazole (Bactrim™) in treating acute exacerbations of chronic bronchitis: summary of European clinical experience. *Ibid.*, pp. 13-14.

Economical b.i.d.

Bactrim™ DS

(160 mg trimethoprim and 800 mg sulfamethoxazole/Roche)

*Due to susceptible organisms. **Please see next page for summary of product information.**

Bactrim™

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin, reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN

Adults. Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children. Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100, Tel-E-Dose® packages of 100; Prescription Paks of 20 and 28. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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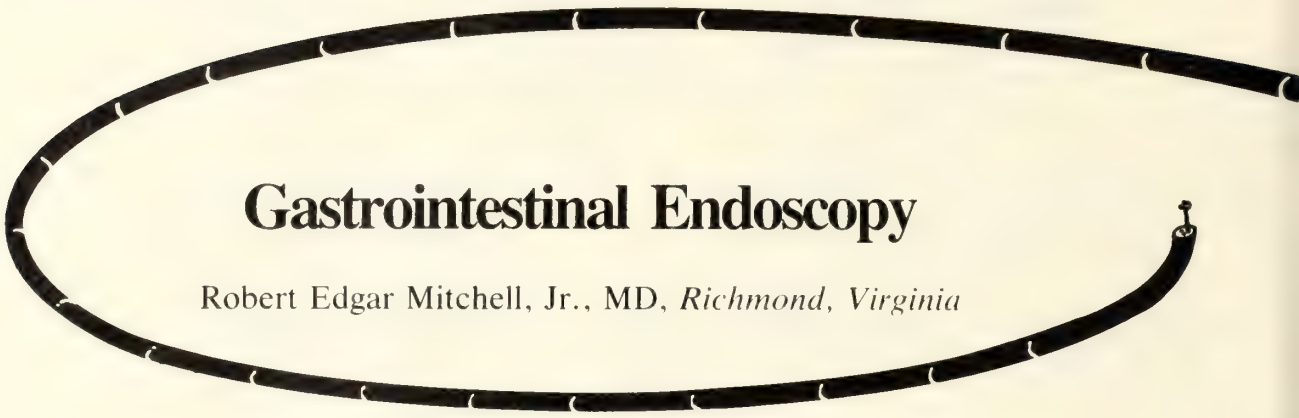

Gastrointestinal Endoscopy

Robert Edgar Mitchell, Jr., MD, *Richmond, Virginia*

After more than 100 years of transition and evolution, gastrointestinal endoscopy has come of age. It has progressed from a straight tube with external illumination and light transmitted by a system of mirrors (for gastroscopy), to the present-day, highly complex, polydirectional, fiberoptic panendoscope designed for either forward or side viewing. The forward-viewing instrument is useful for all of the esophagus, stomach and a portion of the duodenum, as far as the ligament of Treitz. Biopsy and cytology capabilities are present in both forward- and side-viewing endoscopes. The side-viewing endoscope may be most useful in evaluating the ampulla of Vater, as well as enhancing and clarifying other esophageal, gastric or duodenal abnormalities, especially beyond the superior flexure of the duodenum.

The biopsy forceps, which are of the bayonet type, allow for multiple biopsies. Brushings and lavage techniques combine to afford a high yield of accuracy in histopathological diagnosis. Biopsies and brushings may now be supplemented with color photographs taken with an ingenious gastrocamera.

Endoscopic retrograde chloangiopancreatography, (ERCP), utilizes the side viewing endoscope. It has proven to be an excellent procedure for both diagnosis and treatment of pancreatic and biliary tract disorders. It is especially useful in diagnosing, and sometimes removing via papillotomy, common bile duct stones. Thus, we now have another option for poor-risk surgical patients. It allows for cannulation of the ampulla of Vater and pancreatography by the gentle injection of radiopaque material (Renografin-60) under fluoroscopic control to the pancreatic ducts and biliary tract. The introduction of contrast material must be carried out with great care to attempt to avoid acute pancreatitis or cholangitis, which may be very transient and minor or severe. Diagnosis of cancer of the pancreas and chronic pancreatitis, as well as ductal abnormalities, may be made by this technique, including evaluation of the jaundiced patient. In addition, extrahepatic cholestasis may be explained by this procedure.

Endoscopic sphincterotomy (incision of the sphincter of Oddi) may be useful in removing gallstones and tumors of the papilla of Vater. By removing the blockade, pancreatitis may be alleviated to some degree. This procedure is done by means of an electrosurgical wire attached to the endoscope or by actually cutting the sphincter with a tiny device containing a blade.

Even though current experience suggests less danger from endoscopic sphincterotomy in the hands of an experienced endoscopist rather than from general surgery, neither is yet mutually exclusive. Unfortunately, most patients with symptomatic pancreatic cancer are inoperable when first diag-

Address correspondence to Dr. Mitchell at 7601 Forest Avenue, Richmond VA 23229.

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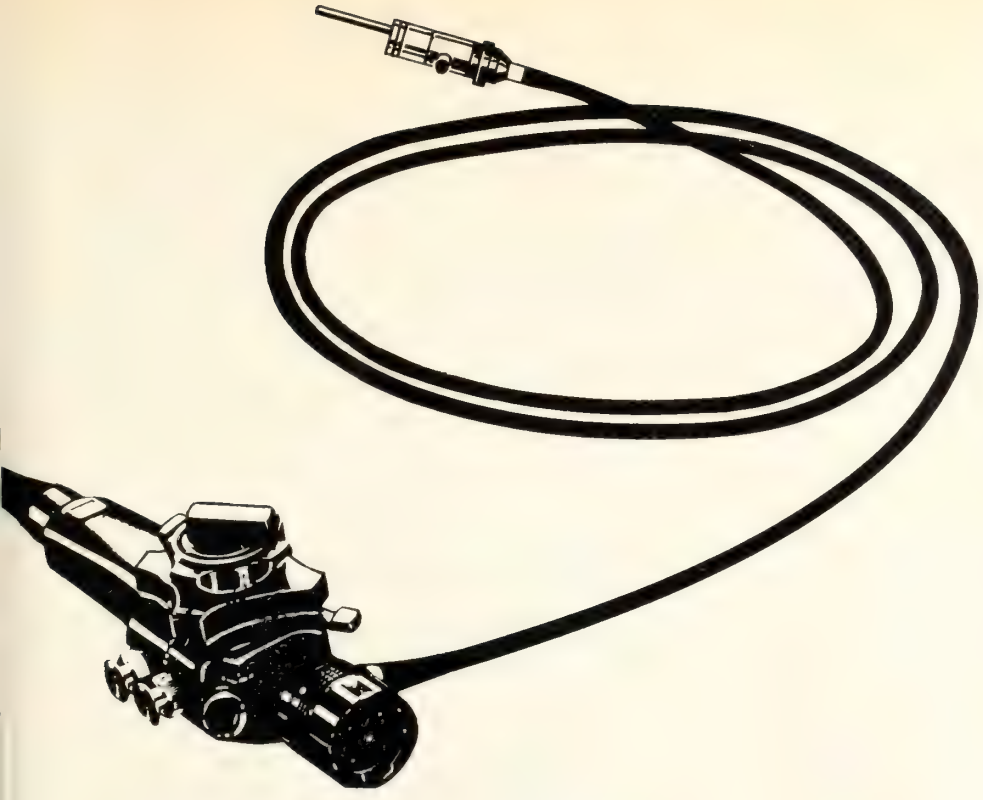

nosed by ERCP. Other uses of ERCP include injection delivery of drugs, placing of catheters into the common bile duct for drainage procedures, delayed pancreatography, and even balloon dilatation of infrastrictures.

Endoscopic sclerotherapy of varices has been increasing in use and is especially helpful in patients who have bled previously and are poor risks surgically. Sclerosants must be carefully selected. Some report the effectiveness of, as well as the damage caused by, the sclerosing agents, and these are directly proportional. Sodium morrhuate, thrombin and ethanolamine oleate have all been used as sclerosants. Laser photocoagulants and electrocautery are being used more in experimental than clinical settings for sclerosing varices, with laser also being used at the present time to sclerose bleeding peptic ulcers, but both offer promise in the control of upper gastrointestinal bleeding, especially after more experience is gained with these techniques. It should be emphasized, however, that mortality from bleeding varices remains high and most agree there is not yet an optimal treatment. The rate of rebleeding, as well as mortality, are both reduced by experienced endoscopists injecting sclerosant material into a varix. We still do not know, however, whether or not this procedure prolongs life.

Fiberoptic sigmoidoscopy and colonoscopy allow for full visualization of the entire colon. The flexible, highly maneuverable instruments currently in use allow for adequate traversing of the sigmoid colon, as well as the splenic and hepatic flexures in most patients. The same capabilities are present as in the esophagogastroduodenoscopes. Biopsies, brushings and lavages are all done, plus photographic documentation of any lesion of the colon. Polypectomies are facilitated by the snare-cautery methodology. Small polyps or malignant lesions which could be missed by roentgenography may be seen with the fiberoptic colonoscope. Dysplasia of the colon and certain cases of inflammatory bowel disease may be diagnosed better with the fibercolonoscope. Chronic blood loss from the lower gastrointestinal tract, unless otherwise explained, probably should be evaluated by fibercolonoscopy.

Laparoscopy (peritoneoscopy) is a very old technique, long popular in Europe, and now regaining popularity with gastrointestinal endoscopists in this country. Under pneumoperitoneum, the laparoscope can directly visualize the liver and other abdominal organs and provides a vehicle for direct biopsy of the liver and other areas. Ascites, hepatomegaly, metastatic disease and localized liver disease are especially helped in diagnosis by this procedure. It should be added that gynecologists find this instrument of tremendous help within their field of endeavor.

Newer techniques utilizing endoscopes are currently underway. One of these is a new type of small bowel endoscope which should facilitate visualization of inaccessible areas. If successful, this

technique would actually allow endoscopic accessibility to virtually all areas of the gastrointestinal tract.

Some studies suggest routine panendoscopy may be 30%–50% more accurate than roentgenography in correctly diagnosing small ulcers or bleeding points. These results should be tempered with the knowledge that we need both disciplines, as things now stand, for completeness. As therapeutic endoscopy becomes more applicable, however, especially with laser photocoagulation usage, the outlook for gastrointestinal bleeding treatment improves. It may well be that five or ten years down the road, laser cautery may make the problem of gastrointestinal tract bleeding a moot one.

The growing sophistication and importance of gastrointestinal endoscopy, starting with the rigid, then the semi-rigid gastroscopes of Schindler (1932), followed by the flexi-rigid esophagoscope (1949), and finally the flexible gastric fiberscopes of Hirschowitz, Curtiss, Peters and Pollard (1957, 1963), have led to our present explosion of interest in these instruments as our knowledge and daring have progressed apace. Present soundings suggest a bright future for gastrointestinal endoscopy. Utilizing such precise procedures and equipment, as well as those yet to come, this rapidly growing field should go far in further explaining gastrointestinal tract pathology, pathophysiology and physiology.

Is the Endoscopist's Eye Exposed to Radiation?

Stephen F. Quinn, MD, and
Hubert A. Shaffer, MD,
Charlottesville, Virginia, and
R. L. Smathers, MD, *St. Louis, Missouri*

FLUOROSCOPIC guidance is frequently used during endoscopic procedures such as bronchoscopy when localization is needed for bronchial brushing and biopsy, or during endoscopic retrograde cholangiopancreatography when adequacy of duct filling must be monitored. Recently, several endoscopists have expressed concern that X-rays might be transmitted through the fiberoptic endoscope, resulting in excessive radiation exposure to their faces and, particularly, to their viewing eyes.

Although the radiation dose received by patients and radiology personnel during various diagnostic procedures has been determined[1,2] we could find no quantitative studies of radiation exposure to the eye of the endoscopist during fluoroscope-assisted endoscopy. We therefore conducted an experiment with two objectives: first, to determine if X-radiation can be transmitted through the fiberoptic endoscope and, second, to quantitate the additional exposure, if any, to the face and eyes of the endoscopist.

Two different fiberoptic endoscopes were evaluated—an Olympus GIF Type D and Olympus BF Type-58. Fluoroscopy was produced with a Philips Diagnost 73 with a 35-cm Scopomat 73-image intensifier field, 100 kW X-ray tube with a 0.6-mm focal spot, and a Maximus M-100 generator at a control setting of 2.2 mA and 94 kVp. Radiation dose from fluoroscopy was measured in millirads (mrad), us-

ing lithium fluoride thermoluminescent dosimetry (TLD). One TLD crystal was placed on the endoscope eyepiece, and another was placed at an equal distance (50 cm) from the X-ray source but was left unprotected from the source.

Three pairs of dose measurements were obtained, each after 10 minutes of fluoroscopy, with the distal 10 cm of the endoscope placed in the fluoroscopic field in different positions relative to the direction of the X-ray beam.

The first pair of measurements, using an Olympus GIF Type D endoscope, was taken with the tip of the endoscope perpendicular to the direction of the X-ray beam. The second pair was obtained with the same endoscope, but with the tip parallel to the X-ray beam and pointed towards the source. The third set of measurements, using an Olympus BF Type-58 endoscope, was taken with the tip of the endoscope parallel to the X-ray beam and pointed towards the source.

The first dose observation, with the endoscope tip perpendicular to the X-ray beam, measured 14.0 mrads/10 min at the unprotected point and 2.0 mrads/10 min at the eyepiece.

The second observation, with the endoscope tip parallel to the X-ray beam, measured 13.0 mrads/10 min at the unprotected point and 1.5 mrads/10 min at the eyepiece.

The third observation, with the endoscope tip again parallel to the X-ray beam, measured 18.5 mrads/10 min at the unprotected point and 1.5 mrads/10 min at the eyepiece.

Our data indicates that fiberoptic material in an endoscope does not transmit X-radiation during fluoroscopy. The endoscopist's viewing eye is actually shielded from scattered radiation by the scope and receives a dose approximately six times less than the dose received by the unprotected eye.

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2. Peters PE, Katz G, Safrany L et al: Radiation exposure in patients undergoing endoscopic retrograde cholangiopancreatography and endoscopic papillotomy. Gastrointest Radiol 3(3):353-355, 1978

From the Department of Radiology, University of Virginia Medical Center, Charlottesville VA 22908 (Dr. Quinn and Dr. Shaffer), and the Abdominal Radiology Section, Mallinckrodt Institute of Radiology, St. Louis, Missouri (Dr. Smathers).

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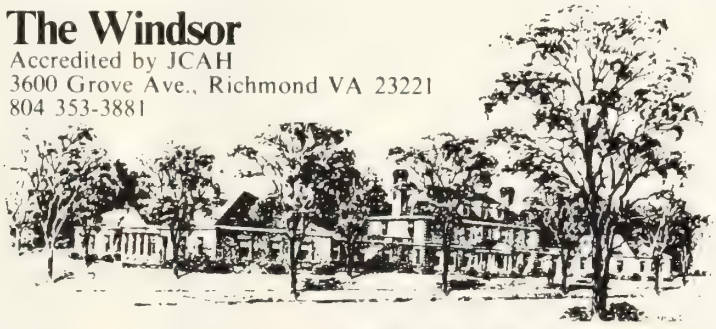

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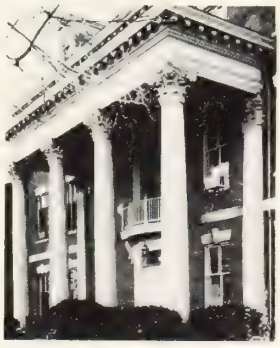

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Clostridal Bacteremia and Necrotizing Colitis with Alcoholism: Case Report

Thomas H. Whitley, Jr., MD,
and Guillermo Pinzon, MD,
Danville, Virginia

If not treated promptly and appropriately, infection with *Clostridium septicum* leads to death in over 70% of patients. Most of the reported cases of *C septicum* bacteremia have been associated with some form of malignant disease, either solid tumors, often of the colon, or hematologic neoplasia.[1] Other reports, however, suggest that states of immunocompetence may predispose to this often fatal infection. It has been reported in association with lesions of non-neoplastic agranulocytosis[2] and with granulocytopenia without malignancy[1], and alcoholism and its complications have been described as precipitating factors for both clostridial infection[3] and leukocyte malfunction.[4]

We report a case of associated *C septicum* infection, necrotizing colitis, and focal cerebral septic hemorrhagic infarction (cerebritis) in an alcoholic patient with pancytopenia and hyperglycemia.

Case Report

A 56-year-old, black, male laborer with a family history of diabetes mellitus and previous hospital treatment for chronic alcoholism was brought to the emergency room following cardiac arrest. He had previously demonstrated euglycemia and normal nemogram. He had not recently been taking drugs, but there was a recent history of heavy alcohol consumption and bloody diarrhea of five days' duration.

Physical examination showed a blood pressure of 60, pulse rate of 120, and a rectal temperature of 39.8°C. He was unresponsive, and manual ventilation was required. The pupils were fixed and dilated, the skin warm and dry. Results of laboratory studies showed a hemoglobin of 7.9 gm%, hematocrit 22%, WBC 3,000/ml and platelet count of 30,000/ml. Differential showed 35% band neutrophils and 44% segmented neutrophils with toxic granulations. Blood glucose was 423 mg/dl, HCO_3 18 mEq/l and urea nitrogen 76 mg/dl. LDH was 1225 u/L, serum GGT 246u/L, ethyl alcohol 0%. Two blood cultures revealed *Clostridium septicum*. Despite vigorous intravenous fluid and respiratory and cardiovascular support with insulin, thiamine, tobramycin and cefamandole, the patient died two hours after admission.

At autopsy, the bone marrow was hypocellular due to a decrease in myeloid elements, with orderly maturation of all series. The entire colon, especially the sigmoid and rectum, was superficially ulcerated and hemorrhagic. Membranes focally covering the surface contained colonies of spore-producing, gram-positive rods (Fig. 1), as did the submucosa. The right cerebral hemisphere revealed a large area of necrosis and hemorrhage with vasculitis and again colonies of spore-producing, gram-positive rods were seen (Fig. 2). Post-mortem culture of blood and involved cerebrum grew *Clostridium* species, toxigenic to mice and typical of the alpha toxin of *C septicum*. The hepatic portal triads revealed early fibrosis, and there was moderate fatty metamorphosis of the hepatocytes. The pancreatic islets were enlarged and hypercellular, with some minimal focal clustering of islets produced by interstitial fibrosis and atrophy of acinar tissue. Diabetic microangiopathy and significant arteriosclerotic disease were not present at necropsy.

Discussion

In our patient as in other reports, the portal of entry of *C septicum* was the colon.

As for the associated alcoholism, a retrospective study identified alcoholism and its complications as precipitating events in 13 out of 47 patients with clostridial bacteremia.[3] Acute enterocolitis commonly follows consumption of large quantities of

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Submitted 5-3-82.

alcohol, and in some persons this reaction may be related to hypersensitivity to congeners of ethanol.[5] Although the hypoplastic bone marrow in our patient could have been secondary to sepsis, the well-developed colonic lesions and history of alcoholism suggest ethanol-induced colitis or leukocyte malfunction as the primary event.[4] Moreover, profound states of immunodeficiency, characterized by a reduction in the functional capacity of Kupffer cells, lymphopenia and reduced IgG, IgM and IgA, follow the chronic ingestion of ethanol and acetaldehyde.[5] The patient's glucose intolerance probably reflected resistance to action of the administered insulin and may have been related to hyperglucagonemia as seen in bacterial infections.[6]

Non-traumatic cerebral infection due to *C septicum* is an unusual complication of this infrequently encountered organism. To the best of our knowledge, it has been reported only once previously.[7]

The authors appreciate the thoughtful comments of Dr. T. J. Moran and the clerical help of Ms. Sandra Curtis.

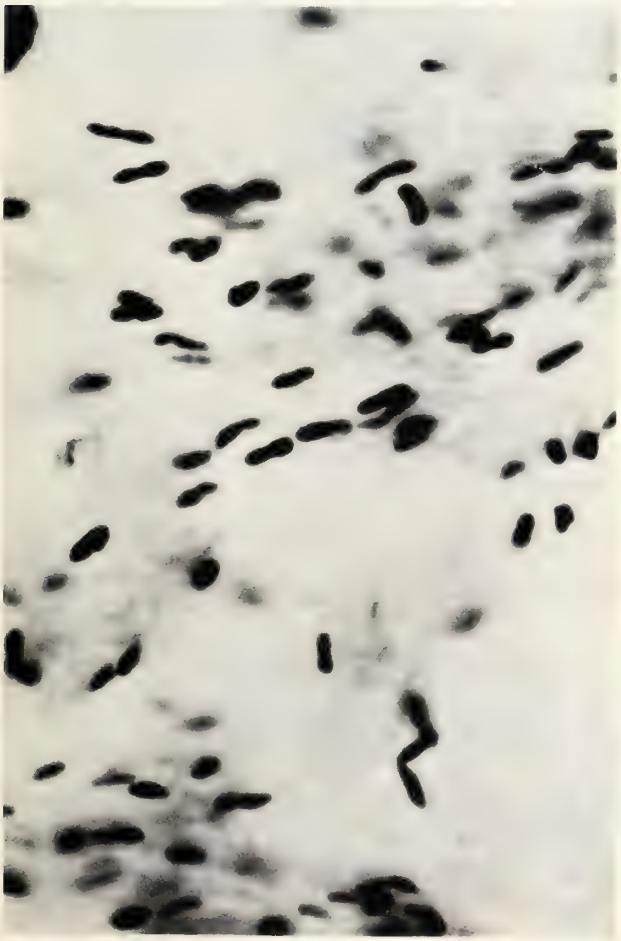

Fig. 1. Mucosal gland of colon loaded with spore-producing gram-positive rods (Gram stain × 1000).

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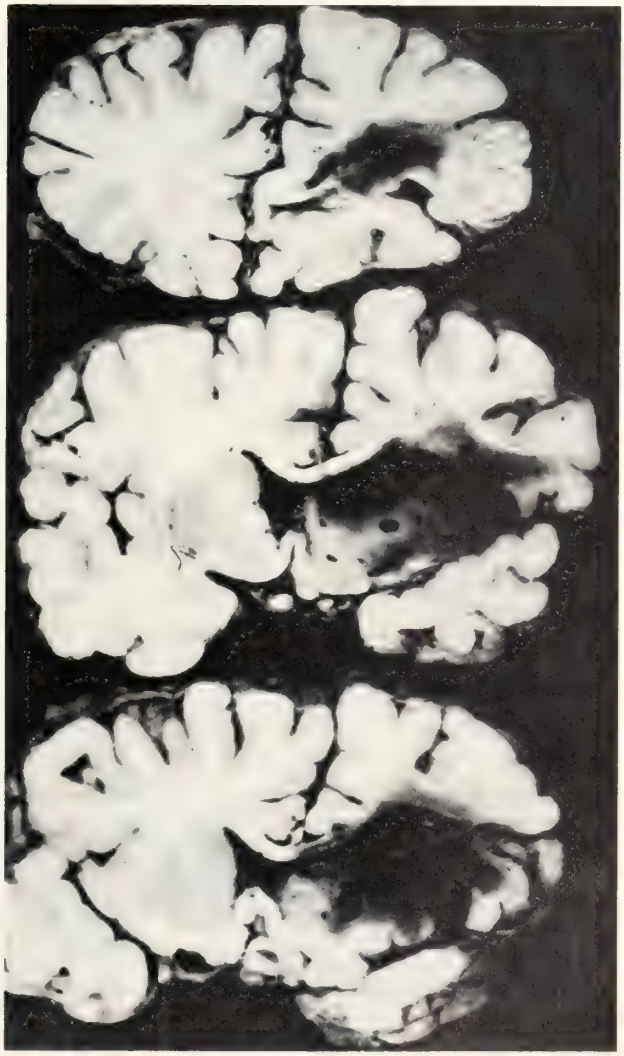

Fig. 2. Gross photograph of cerebrum with septic hemorrhagic infarction of right hemisphere.

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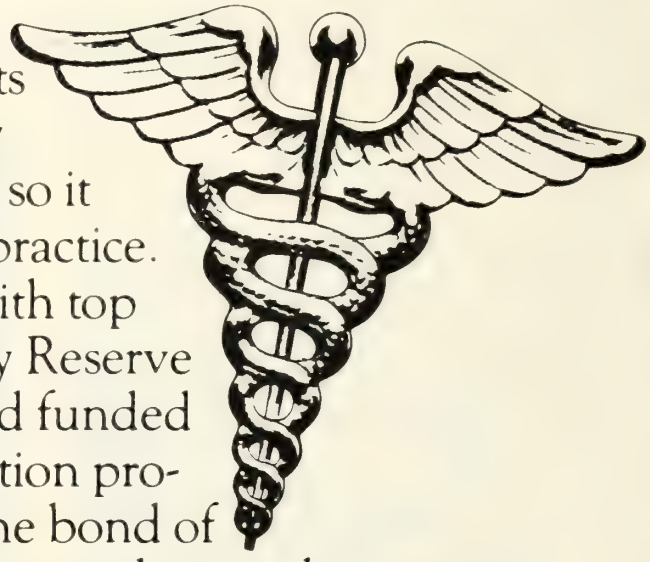

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Use and Abuse of Adrenal Corticosteroids

Robert D. Lovinger, MD,
Richmond, Virginia

THE adrenal cortex comprises three functional zones.[1] The outermost zone produces aldosterone, a steroid with mineralocorticoid activity which mediates sodium balance and extracellular fluid volume by increasing sodium reabsorption from the renal tubule and other parts of the body in exchange for potassium and hydrogen. Aldosterone is regulated primarily by renin via angiotensin and, to a lesser extent, by elevated blood potassium concentration and/or decreased blood sodium concentrations.

The middle zone of the adrenal cortex secretes glucocorticoids, mainly cortisol, which is the principal secretory product of the adrenal cortex. The glucocorticoids protect the body against stress, possibly through maintenance of vascular reactivity to catecholamines, as well as by elevating blood glucose, increasing protein catabolism, and regulating the glomerular filtration rate to control water excretion. Glucocorticoid secretion in turn is controlled principally by pituitary ACTH.

The innermost zone of the adrenal cortex synthesizes sex hormone precursors (primarily weak androgens), which effect the production of sexual hair but have only minor influence on sexual function. The adrenal sex steroids are controlled by a postulated, yet undetermined, pituitary factor, adrenal androgen-stimulating hormone (AASH), and/or ACTH.

Although the naturally occurring glucocorticoids have lesser mineralocorticoid effects and the mineralocorticoids may manifest glucocorticoid properties, each is usually classified according to its major physiologic effect. These hormones do not act immediately because, as steroids, their operative mechanism is through the slow induction of enzymes after direct interaction with cellular DNA rather than with rapid-acting, cell-surface, cyclic, AMP-mediated receptors.

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Submitted 8-19-82.

Corticosteroids

Many naturally occurring and synthetic corticosteroids are presently in use, either for physiologic replacement of endogenous cortisol when production is deficient (as in Addison's disease or adrenogenital syndrome) or often in pharmacologic doses, for their anti-inflammatory properties.[2,3]

Long-term physiologic glucocorticoid replacement usually has no side effect; however, treatment of a patient with pharmacologic doses for allergic, inflammatory, or malignant disease often inhibits growth and elicits cushingoid features, as well as such well-known side effects as osteoporosis, peptic ulcerations, hypocalcemia, pseudotumor cerebri, decreased resistance to infection, diabetes, edema, hypertension, myopathy, mood alterations, and cataracts, increased intraocular pressure or other ophthalmological complications.[4] Moreover, pharmacologic doses of glucocorticoids directly suppress pituitary-stimulated ACTH production and therefore can cause adrenocortical atrophy in proportion to the potency of the corticosteroid and the duration of its administration.[5]

Corticoids fall into two general categories: 1) cortisol, a naturally occurring compound that fits into the normal, cellular, corticosteroid receptor, and 2) all other natural and synthetic steroids which must be somehow altered to fit into the corticosteroid receptor before their biologic action can be expressed. Thus, when rapid onset of action is demanded, cortisol is the glucocorticoid of choice. Although a single dose of corticosteroids, even a large one, is virtually without harmful effects and even high-dose therapy over a few days is unlikely to produce harmful results in the absence of specific complications or extreme doses, as corticosteroid therapy is prolonged or exceeds the equivalent of

simple substitution, the incidence of disabling and potentially lethal effects increases proportionately.[3] Accordingly, the abrupt cessation of prolonged high-dose corticosteroid therapy may create a significant risk of a potentially life-threatening adrenal insufficiency and must be adjusted and reevaluated periodically for the best therapeutic effect for the stage and activity of the particular disorder. Most "equivalent dosages" of adrenal steroids are based on their anti-inflammatory or other non-endocrinologic properties in adults; however, in pediatric application, when the physiologic property considered is growth, the steroid preparation customarily used may be more potent than is desirable, e.g., prednisone may be four times as potent as cortisol in its anti-inflammatory effects but up to eight times as potent in its growth suppression action.[2]

Maintenance. Under basal conditions the adrenal gland secretes 12 mgs (± 3 mg) of cortisol per square meter of body surface area per 24 hours.[6,7] Because of decreased absorption, the dose of oral preparations must be double that of parenterally administered steroids, i.e., 25 mg/M^2/day. Steroids with short half-lives, i.e., oral cortisone acetate, should be given in at least three divided doses per day, whereas longer-acting oral preparations, i.e., prednisone, desamethasone, can be used twice a day.

Stress. In response to such stress as fever exceeding 101°F, anesthesia, etc., the normal adrenal gland secretes three to 15 times the basal amount of cortisol, so that the stress dose of exogenous steroids must be at least three times the basal dose, i.e., 45 mg/M^2/day parenterally or 75 mg/M^2/day orally, for the duration of the acute stress.[8] In an adrenal crisis, intravenous hydrocortisone sodium succinate (Solu-Cortef®) should be administered as indicated for preparation for surgery.

Preparation for Surgery. At 12-hour intervals beginning 36-48 hours prior to surgery, a stress dose of cortisone acetate (45 mg/M^2) can be administered intramuscularly until surgery so that its 18-hour peak activity and 48-72-hour effectiveness are cumulatively reinforced. Alternatively, intravenous hydrocortisone sodium succinate (Solu-Cortef®) in stress doses (45 mg./M^2) can be given at anaesthesia and another 100 mg/M^2/day given by continuous infusion. Postoperatively the stress dose should be maintained for two to three days, until maintenance therapy can be resumed.

Pharmacologic Administration. Because no uniform guidelines are currently available for pharmacologic administration of steroids in chronic inflammatory disease states, the following empirical guidelines are offered: prednisone or prednisolone, 1-2 mg/kg-day in 2-4 divided doses; dexamethasone, 0.1-0.2 mg/kg/day in 2-4 divided doses. During viral infections the dose should be reduced to stress levels.[9] Tuberculin skin-testing is a prerequisite to chronic pharmacologic corticosteroid therapy.

CORTICOSTEROID THERAPY

Maintenance:	Parenteral—12 mg (±3)M^{2*}/day
	Oral—25 mg/M^{2*}/day
Stress:	Parenteral—45 mg/M^{2*}/day
	Oral—75 mg/M^{2*}/day

Preparation for Surgery: 1) If starting 36-48 hrs prior to surgery, use cortisone acetate 45 mg/M^{2*} every 12 hrs IM. 2) If starting immediately prior to surgery, use Solu-Cortef® 45 mg/M^{2*}/IV push at time of anesthesia and 100 mg/M^{2*}/day given by continuous infusion.

* When height and weight are normal, seven pounds (as in an infant) is about 0.22M^2, 60 pounds is about 1M^2, and 160 pounds is about 1.75M^2.

Withdrawal of Glucocorticoids. Withdrawal of stress or pharmacologic doses of glucocorticoids from patients so treated for more than 14 days must be gradual. The steroid dose should be decreased by half every 3-4 days until physiologic replacement-dose levels are reached (25 mg/M^2/day orally); thereafter the dose should be halved every two weeks or decreased by an equivalent of 5 mg of cortisol each week. Any patient who has received pharmacologic doses of glucocorticoids for more than 14 days within a year requires coverage during subsequent times of stress at a dose of 75 mg/M^2/day orally of cortisol or its equivalent.[10]

Because it is difficult to reduce pharmacologic dosages of prednisone and/or dexamethasone in small graduations by the preceding schedule, it is often easier to wean patients off these dosages by first converting them to equivalent doses of cortisone acetate given three times a day and then reducing the dosages.

During withdrawal of steroids, patients must be watched for symptoms of adrenocortical insufficiency (weakness, muscle pain, tinitis, hypoglycemia, hypotension), and, if any become overt, it may be necessary to increase the dosage and reduce it more slowly.

Mineralocorticoids

Since mineralocorticoids affect sodium retention, a deficiency leads to hyponatremia, hyperkalemia, hypovolemia, hypotension, and weakness. It can be treated either by deoxycorticosterone acetate

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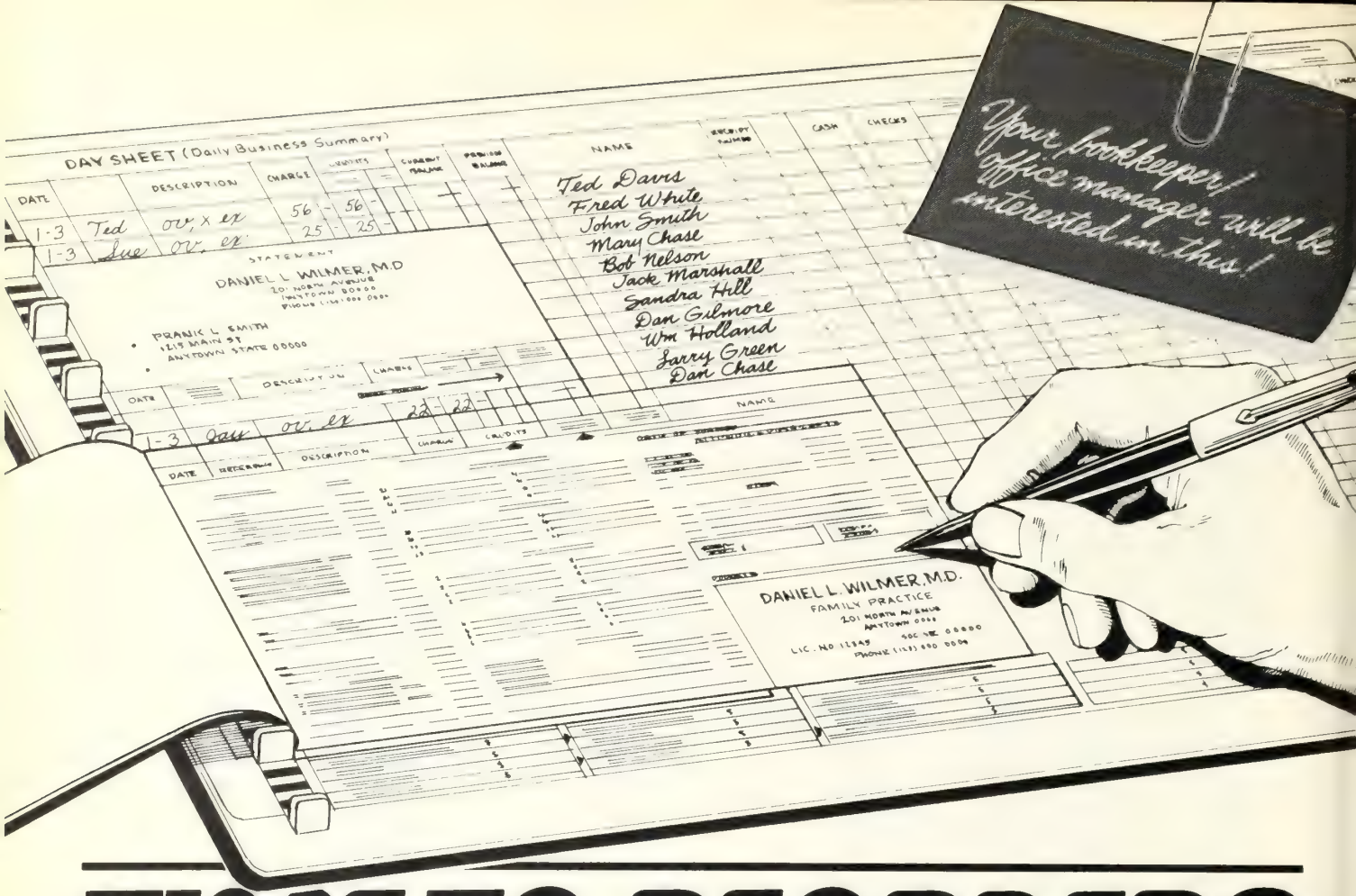

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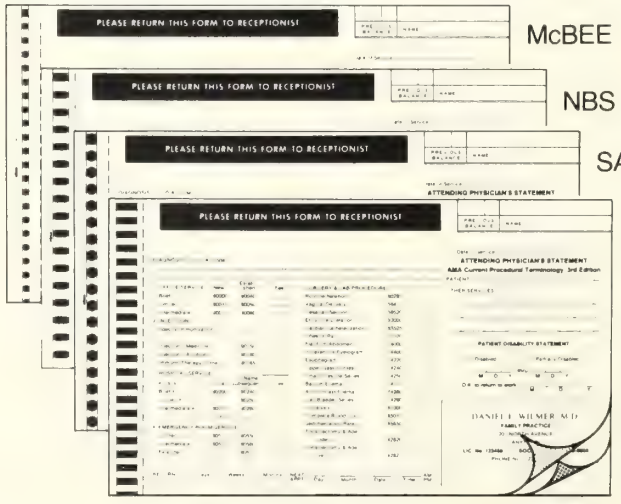

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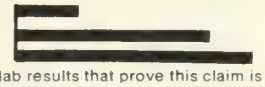

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VIRGINIA MEDICAL

EDITORIAL

The Medical Scientist: An Endangered Species?

THE POTENTIAL for scientific discovery in medicine today is boundless. Reports of advances in medical care are made daily throughout our country. The development of the new hepatitis vaccine typifies these miracles of modern medicine. This newly developed vaccine may eliminate the 200,000 cases of hepatitis that occur annually among hospital employees. Moreover, a new treatment for the serious blood disorder thalassemia has been tried successfully, utilizing a drug that activates a dormant gene. It has been suggested that this drug acted at least partly by helping to remove chemical subunits, called methyl groups, from close association with the dormant genes. This clinical application demonstrates conclusively that the discipline of genetic engineering has come to the patient's bedside.

The problem of isolating and synthesizing the growth hormone produced in the hypothalmus recently was solved. Scientists can now start the process of determining how to use this hormone to solve growth disturbances, treat forms of diabetes and speed the healing of wounds and bones.

A unique family of microbes, called prions, has been identified and is implicated in a large number of neurologic diseases, including senility, multiple sclerosis, Parkinson's disease and amyotrophic lateral sclerosis. Prions are too small to be seen through a microscope but can be identified by electrochemical analyses. While the prion has not yet been purified and deciphered, that should be a straightforward task.

Scientists have also made remarkable progress in discovering the etiology of some forms of cancer. Cell geneticists, studying chromosomes into which genes are packaged, have begun to associate specific abnormalities in genes with specific types of cancer. In addition, virologists have clarified the roles that viruses play in altering genes, which may lead to cancer. Finally, molecular biologists have used new tools of genetic engineering to pinpoint precisely which gene, out of tens of thousands present in every human cell, is responsible for causing cancer.

While these scientific advances are remarkable, they account for only a fraction of the promising discoveries that may lead to new therapies to prevent and treat illnesses as well as injuries. The mood in medicine is exciting with the realization that the speed with which scientific discovery is being made is literally explosive.

The miracles of modern medicine are due to a long-term commitment of monies from the federal government to support medical research and the education of health professionals. Over the last 20 years, our government has viewed health education

Originally presented at a joint meeting of the National Advisory Council on Health Professions and the National Advisory Council on Nurses Training of the United States Department of Health and Human Services on January 17, 1983, at Hyattsville, Maryland.

and research as a natural resource and has provided sufficient grants to promote these advances in health care. But just as scientists are making these great strides, federal financing is being restricted. Budget projections for the National Institutes of Health indicate that future allotments will not be commensurate with anticipated inflation, and in real-dollar support, a 25% decrease may occur by 1986.

Federal funds for the education of health professionals are also being dramatically reduced. Given this reduction and the increased cost of training, it is possible that only the well-to-do will be able to pursue a career in medicine. Those who can afford it or who incur large debts to finance it will probably have to pursue private practice rather than a career in research or teaching.

Medicine's greatest energy source is the mind-power generated by our universities and medical schools. Our teachers have within their grasp the means to affect eternity. Their research is and will be our nation's heritage. If federal and state support for medical research and the education of health professionals continues to be restricted, the medical scientist will become an endangered species.

RICHARD F. EDLICH, MD

Department of Plastic and Maxillofacial Surgery
University of Virginia Medical Center
Charlottesville VA 22908

A Golden Opportunity?

MUCH as the buzz word of the 1970s was health maintenance organization, the 1980s will be the decade of the preferred provider organization. Both schemes are stop-gap mechanisms to control spiraling health care costs.

The HMO concept, however, was and is being promoted by the federal government, while the PPO initiative is coming from the private sector, as described elsewhere in this issue.

Also, the HMO financing scheme reduces costs by putting the HMO and its doctors at risk. It also markedly reduces freedom of choice of patients.

The PPO scheme, on the other hand, reduces costs through physicians' agreements to render services at discounted fees, or through reduction of unnecessary utilization.

At this writing there are about 11 million patients covered under HMO plans, which number includes 4 million in the pioneering Kaiser-Permanente Plan. The Kaiser Plan has been profitable and continues to grow, though more slowly than expected. Many other HMOs continue to operate, but several have failed, leaving bewildered patients and confused and frustrated doctors in their wakes.

Preliminary reports show encouraging results for PPOs, i.e., decreased cost of care, increased patient acceptance, and increased census for the participating hospitals. Patient acceptance is enhanced because of wider choice of physicians and hospitals. Out-of-pocket costs to the patient varies according to the PPO but are not large.

The key to success of the preferred provider organization, however, revolves about a broad and enlighted physician participation with acceptance of rigid utilization guidelines and effective peer review. And there is a special problem with that acceptance. The very people who gave us first-dollar coverage and cost-plus hospital reimbursement, and the resulting excessive utilization of services and overbuilding of hospitals, are in many cases the same people who now propose the constricting PPO mechanism. A certain credibility gap exists, and so, in some areas, doctors and hospitals are organizing their own PPOs.

Why should a physician join a preferred provider organization? 1) It enables a fee-for-service practitioner to compete with HMOs. 2) There is a potential for increased patient flow. 3) Rapid claims administration is possible. 4) Financial risk is zero or minimal.

Business, government, and insurance interests are on the move. All feel the medical delivery system needs reform and are working for change through financial schemes. They are, however, still looking to physicians for leadership, and the PPO opportunity to cooperate not only with business but with government, insurance, hospital and consumer interests during the 1980s may represent the last chance we have to preserve the best health care system the world has ever known. The alternative could be an intolerable loss of freedom for doctors and patients alike.

EDWARD A. ZAKAIB, MD

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HISTORICAL NOTES

A Vanishing Tribe: The Monacan Indians of Virginia

In Amherst County, in the eastern foothills of the Blue Ridge Mountains of Virginia, live the descendants of one of Virginia's original Indian tribes, the Monacans. There aren't many of them left, perhaps 300. I came to know some of them through providing pediatric care for their children, and I heard the local lore about them. For instance, in Lynchburg, where these Monacan descendants go for hospitalization, there has been speculation that because of intermarriage their children are prone to birth defects. I investigated this rumor, concluding that it is not true, and was led into an acquaintance with Monacan history.

By the time the English arrived in Virginia, the Monacan Indians had long lived in the Piedmont region of Virginia. Capt. John Smith was the first colonist of record to learn of the tribe; in 1608 he sent 120 men up the James River to find them, and Smith's map of Virginia, drawn in 1612, shows the five Monacan settlements that appear on the map with this article.

Sixty years later, John Lederer, German surgeon and explorer, reported that he visited the Monacans during an expedition into mid-Virginia.

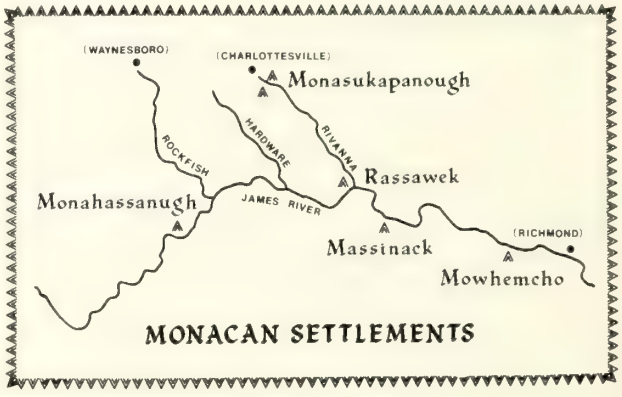
But the colonists were given little opportunity to know the tribe; the Monacans moved about a lot and appear to have been indifferent to the English, so that early chronicles provide only evanescent glimpses of them. It was not until 1894 that historians began putting together, from various records, the bits and pieces of Monacan history.[1,2]

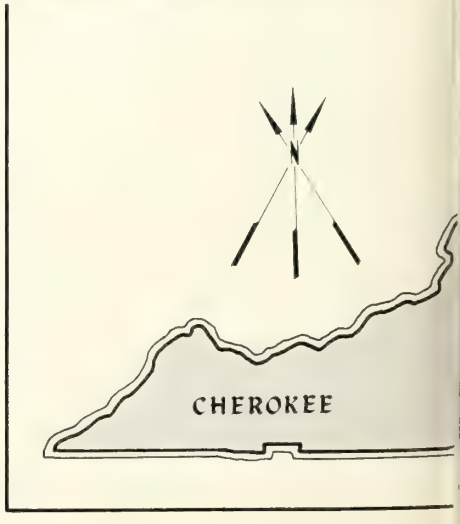
The accompanying map locates the Indian tribes in 17th Century Virginia. The Powhatan Indians belonged to the Algonquin nation. The Nottoway, Meherrin and Cherokee tribes were members of the Iroquois nation. The Monacan, Manahoac, Saponi, Nahyssan, Tutelo and Occaneechi tribes comprised the eastern link of the large Siouan confederacy that extended into the western plains.

The Monacans fell on hard times early in Virginia's history. Beginning in 1680, their enemies, the Iroquois, mauled them with repeated attacks. In the early 1700s the westward expansion of the English began exerting severe pressures. Weakened, the Monacans fell prey to disease and alcohol.

In 1728, a large group of Monacan survivors joined forces with Tuscarora Indians of North Carolina and moved north to settle among the Iroquois. The last trace of these migrant Monacans, known as the Sapponis, died out a century ago.

A small knot of Monacans stayed stubbornly on in Amherst County.[3,4] When, in the mid-1700s, the first white settlers probed the settlement, white men married Indian women. These new alliances abandoned the Indian identity and assumed English surnames. Three surnames were primarily associat-





With the advice and counsel of James W. Hollomon, Jr., of the Jamestown-Yorktown Foundation, these maps were adapted from *Indians in the 17th Century*, by Ben C. McCary, and *Virginia Indians Before and After Jamestown*, by McCary and Parke Rouse, Jr., both published by the Jamestown Foundation.

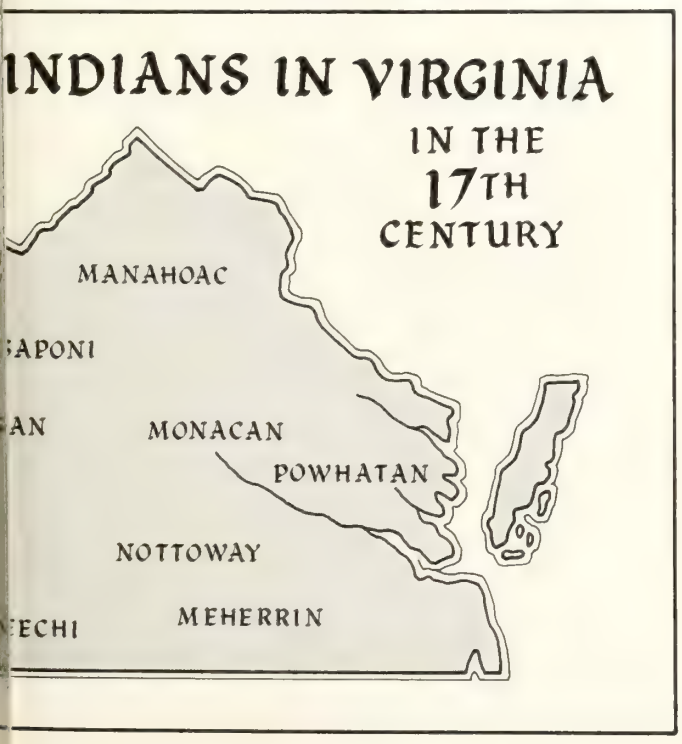
ed with these mixed marriages; I shall here refer to them as the X, Y and Z families.

The Monacan community bought 400 acres of land, the only Indians in Virginia ever to make such a purchase, and created a self-imposed reservation. To this sequestered colony, with its racially mixed families, the racial tensions of the 1800s brought social isolation, and its people began intermarrying. Over the years it became a common occurrence for first cousins to marry.

There also were some marriages with freed slaves, but those who married blacks were later ostracized from the colony, and the remaining white-Indian group refused to associate with blacks.

Early in the 20th century an Episcopal mission with an elementary school for the children was established in the community. The Monacan descendants welcomed its support and, although they were encouraged by mission workers to find roles in American society, tended to become even further isolated and thus did not participate in the benefits of social progress of the 1950s. Their children were not admitted to public schools until after the integration of blacks had taken place, and it was not until 1972 that one of the community's youngsters was graduated from high school.

Today the Monacan descendants in Amherst County are living and marrying outside of their previously insular society. Most of those I have talked with know little of their heritage, beyond their grandparents. Some are curious about it, others want to forget. Their survival is a tribute to adaptation, but it is sad to see this near-extinction of the last Monacan identity.



To study the incidence of birth defects in the Monacan community, I reviewed the medical records of the births and childhood admissions of children with the surname X or Y at Lynchburg's two hospitals, Virginia Baptist and Lynchburg General, for the years 1945–1982. It should be noted that these records are incomplete, for several reasons: 1) Until ten years ago, Indian descendants were labeled "Negro" on their birth certificates; because of this, Indian mothers spurned hospital deliveries. 2) In any case, many of the mothers preferred home deliveries. 3) Some mothers may have gone to other hospitals to deliver their babies.

The records yielded 67 newborn with the surname X. Four of these were born with congenital defects, one each with meningocele and cleft lip and two with defective hearts. Against a norm of 2.4%[5], I found in these 67 infants a 6.0% rate of congenital birth defects. There were eight cases of prematurity among the total 67 newborn, which gave a prematurity rate of 11.9% against a norm of 7.0%[6].

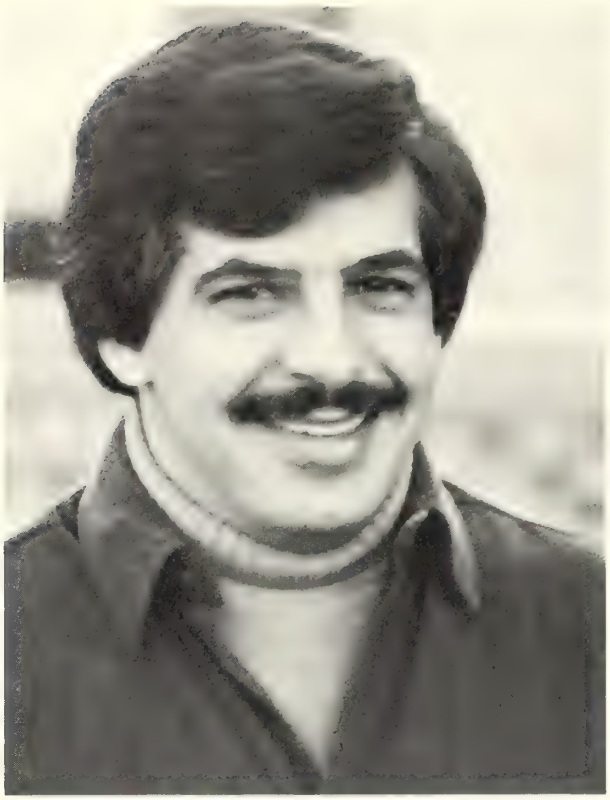
The hospital records showed the births of 50 infants of the Y family. In contrast to the newborn of family X, the records of these 50 Y-family babies showed not a single birth defect or premature delivery.

Since the Indian gene in both the X and Y families came in all likelihood from a common Monacan tribe, one could speculate that the increased percentage of birth defects in the X family might be due to the white genotype. When one figures the ratio of all 117 cases from both families, the percentage of birth defects is close to the norm.

It should be noted that when I next examined the records for childhood admissions of members of the X and Y families, I found one case of amytonia congenita, diagnosed in a 9-month-old child of the Y family, but since I had limited my study to birth defects, it is not incorporated into the foregoing figures.

Generally, the childhood admissions from the families X and Y did not show a preponderance of any particular abnormality or disease, which surprised me, since susceptibility to tuberculosis, smallpox and otitis media has been reported in other Indian cultures, and in Virginia the Chickahominy Indians have shown an increased incidence of cystinuria associated with renal stones.[7]

In addition to the case of amytonia congenita



A Monacan descendant today. *Photograph by the author.*

described above, the childhood admissions of the X- and Y-family Monacan descendants showed three cases of pneumonia, two of malnutrition, and one each of asthmatic bronchitis, eczema, Reye's syndrome, stomatitis, pharyngitis, cervical adenitis, laryngotracheobronchitis, poliomyelitis and diarrhea.

PETER W. HOUCK, MD

1901 Thomson Drive
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Richard W. Wingfield, MD

Dr. Richard W. Wingfield, longtime family physician in Keller, Virginia, died at Northampton-Accomack Memorial Hospital on January 7 at the age of 57.

A native of Elkins, West Virginia, Dr. Wingfield was graduated from the Medical College of Virginia in 1952 and received training at Edward W. Sparrow Hospital, Lansing, Michigan. He served in World War II and the Korean War and practiced in West Virginia before moving to the Eastern Shore in 1966.

Dr. Wingfield was a member of the Accomack County Medical Society and The Medical Society of Virginia for 27 years.

Memoir of June C. Shafer 1917–1983

By John E. Alexander, MD, James M. Moss, MD, and James P. Rotchford, MD

We were greatly saddened by the sudden death on January 16 of Dr. June Carol Shafer. Not only have we lost a talented and hightly respected physician but also a warm friend who will be missed by her colleagues and her patients.

Dr. Shafer was born in Chicago and received her MD degree from the University of Wisconsin in 1937. In 1942–1945 she was the first woman resident in dermatology at the University of Virginia Hospital. The late Dr. D. C. Smith, chairman of the Department of Dermatology, stated "She was the best resident that I ever had." After two years on the faculty of Columbia University she opened her office in Arlington, Virginia, and became a consultant in dermatology at Walter Reed Army Hospital in 1947. In 1970 she was promoted to emeritus consultant. She joined the faculty of Georgetown University in 1956 and was promoted to clinical associate professor of dermatology. She served as assistant professor of dermatology at Johns Hopkins University from 1971 to 1979.

Dr. Shafer was a recognized authority on the skin changes produced by radiation and frostbite. She was the second woman to be elected to the American Dermatologic Association and the first woman to serve on the American Board of Dermatology. She had over twenty publications and major scientific presentations. She was one of the first fellows of the American College of Physicians in Northern Virginia and sponsored many young physicians in that organization.

Those of us who were privileged to know and work with Dr. Shafer were associated with a superior person who combined scientific achievement with a deep love and respect for her patients. Her passing is a great loss to the medical community.

Alexander T. Mayo, MD

Dr. Alexander T. Mayo, 77 years old, died February 24 in a hospital in Portsmouth, where he was a family practitioner from 1930 until his retirement in 1979. A native of Portsmouth, Dr. Mayo was a graduate of the University of Virginia and trained at Roanoke Memorial Hospital. He had been chief of staff of Portsmouth General Hospital.

A member for 40 years of the Portsmouth Academy of Medicine and The Medical Society of Virginia, Dr. Mayo also belonged to the American Academy of Family Physicians, the American Society of Abdominal Surgeons and the Industrial Medical Association.

WHO'S WHO

For a long time medicine and camellias played tug of war with **Dr. James M. Habel, Jr.,** of Suffolk, but finally, early this year, the camellias won. Dr. Habel saw his last patient at the Lakeview Clinic and performed his last surgical procedure at Louise Obici Memorial Hospital, then turned full-time attention to his camellias.

Raising camellias has been a major preoccupation of the obstetrician-gynecologist since 1950, when he added a greenhouse to his home. The camellia population quickly outgrew that structure, so a second, larger greenhouse was built. When, recently, the flowers again pressured for space, the first greenhouse sprouted an addition.

These are prize-winning camellias, mind you. Dr. Habel is a past president of the American Camellia Society and a trustee of the American Camellia Foundation, and his camellias measure up to their grower's horticultural status. The interior of the Habel home looks like a silver vault, with wall-to-wall trophies, and now that her husband is concentrating single-mindedly on the camellia circuit, Mrs. Habel is braced for a fresh onslaught of awards.

At 73, Dr. Habel rivals his camellias in vigor. He was born in Amelia County, where his father was a country doctor. "He also owned a drug store, and I almost went into pharmacy," the son remembers, but after graduation from the College of William and Mary, he went on to an MD at the Medical College of Virginia.

First he practiced in Franklin, then in 1940 he went to Suffolk, where for almost a decade he was the area's only obstetrician. He delivered so many babies he couldn't keep count. "For a long time 300 or more a year," he estimates and adds, "I've had third-generation births." The oldest pregnant patient he dealt with was 52 years old; the youngest, 12 years.

Both of Dr. Habel's daughters married physicians: Alie-Blue is married to **Dr. William C. Everett, Jr.,** Newport News, and Sue is the wife of **Dr. Stephen L. Green,** Hampton.

Dr. Bertram L. Warren, Jr., Virginia Beach, has been elected chairman of the Virginia Section of the American College of Obstetricians and Gynecologists for a three-year term. Dr. Warren is president elect of the Virginia Beach Medical Society.

Appointed by Secretary Richard S. Schweiker of the Department of Health and Human Services to the National Advisory Council of the National Health Service Corps was **Dr. Frank S. Royal**, Richmond general practitioner and president of the National Medical Association.

Drs. Howard and Georgeanna Jones of Norfolk, founders of the nation's first in vitro fertilization clinic, were the recipients of the first health care award of the Association of Virginia Planned Parenthood Associates given late last year. Another honor was bestowed on Dr. Georgeanna Jones by the James Madison University Faculty Women's Caucus, which named her Outstanding Woman of Virginia.

Authorities of Turkish Cyprus have given 20 pieces of ancient

Dr. Habel operating on his retirement hedge.
Photo by Harvey White

Cypriot pottery, some of it more than 3,000 years old, to the University of Virginia in honor of **Dr. Norman J. Knorr**, dean of the School of Medicine, and **Dr. Vamik D. Volkan**, the Cyprus-born director of the university's Blue Ridge Hospital division. The gift recognized the two doctors' contributions in establishing mental health services on the Turkish side of the war-torn Mediteranean island.

Dr. William F. Murphy, Jr., Norfolk pediatrician, received the 1982 Outstanding Child Advocate Award from the State Division of Children. The honor recognized Dr. Murphy's leadership in developing a cardiac screening program for Tidewater school children and a similar program of national scope.

Dr. Edgar N. Weaver, Roanoke, and Senator Harry F. Byrd, Jr., have been elected to the board of trustees of the University of Virginia's Medical School Foundation, which administers funds raised by the university's medical alumni.

Mining **Dr. Mary Elizabeth Johnston Brittain**'s memories of medical practice in 1934, when she was a fledgling physician in Tazewell, a newspaper reporter in Southwest Virginia generated a lively feature story for a recent issue of the Clinch Valley News.

Commenting on the many medical changes she has seen, "Dr. Mary" observed that there are now "a lot of drugs that are a lot harder to spell and have a lot more reactions, but used with discretion, they are a lot more effective."

Dr. Mary's father, Dr. P. D. Johnston, was a Tazewell GP before her; she joined him in practice after graduation from the Medical College of Virginia and internship at Johnston-Willis Hospital in Richmond.

The joint Johnston practice, she recalls, was "75% house calls and 25% office visits. One of us was in the office at all times, and one of us was on the road at all times." That changed during World War II, when stamps for rationed gas were alloted for trips to the doctor. Also, more and more equipment became available for office use, giving impetus to office treatment.

Of conditioning her father's patients to being treated by a female doctor she remembers, "I didn't really have any problems. When my father wasn't available, they were glad enough to get me."

Shortly after she retired in 1978, Dr. Mary married another retired Tazewell GP, **Dr. Rufus Brittain,** who had been widowed by his wife's death in an auto accident. Both Dr. Mary and Dr. Rufus are veteran members of both the Tazewell County Medical Society and The Medical Society of Virginia, and Dr. Rufus has been president of the Virginia Academy of Family Physicians.

Partnership of veterans: Dr. Mary and Dr. Rufus.
Photo by Leesa Wood for the Clinch Valley News

New president of the Richmond Memorial Hospital staff is **Dr. Walton M. Belle** of Richmond, general surgeon.

The medical staff of Winchester Memorial Hospital recently elected as its president **Dr. Herbert A. Wood, Jr.,** Winchester internist.

Dr. Robert J. Karmy, Woodstock, is the new president of the staff of Shenandoah County Memorial Hospital.

In Danville, **Dr. M. D. Richmond** was named a trustee of Memorial Hospital of Martinsville and Henry County.

Dr. F. Elliott Oglesby, Richmond, is the newly elected chief of staff of Richmond's Stuart Circle Hospital.

Dr. Jerome D. Gorman, Richmond, has been elected a diplomate of the American Board of Emergency Medicine, and **Dr. Fritz H. Andersen,** Annandale, has been elected a fellow of the American College of Cardiology.

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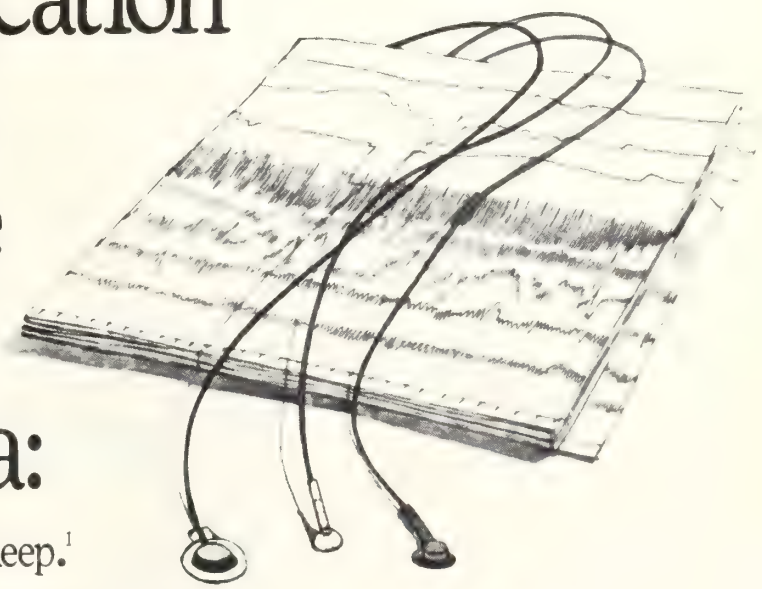
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References: 1. Kales A *et al: J Clin Pharmacol 17*:207-213, Apr 1977 and data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Kales A: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **3.** Zimmerman AM: *Curr Ther Res 13*:18-22, Jan 1971. **4.** Kales A *et al: JAMA 241*:1692-1695, Apr 20, 1979. **5.** Kales A, Scharf MB, Kales JD: *Science 201*:1039-1041, Sep 15, 1978. **6.** Kales A *et al: Clin Pharmacol Ther 19*:576-583, May 1976. **7.** Kales A, Kales JD: *Pharmacol Physicians 4*:1-6, Sep 1970. **8.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **9.** Dement WC *et al: Behav Med 5*:25-31, Oct 1978. **10.** Vogel GW: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **11.** Karacan I, Williams RL, Smith JR: The sleep laboratory in the investigation of sleep and sleep disturbances. Scientific exhibit at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971. **12.** Pollak CP, McGregor PA, Weitzman ED: The effects of flurazepam on daytime sleep after acute sleep-wake cycle reversal. Presented at the 15th annual meeting of the Assc[illegible] for Psychophysiological Study of Sleep, Edinb[illegible] 30-July 4, 1975. **13.** Data on file, Hc[illegible] Nutley, NJ.

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Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

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Manager
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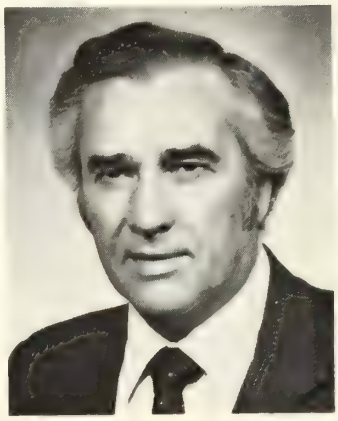

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New vaccine at the ready in rabies risk

Glenn H. Shepard, MD, and Daryl J. Hodgkinson, MD, both of Newport News, Virginia; Jeffrey L. Lake, MS, staff epidemiologist, Virginia Department of Health; and Managing Editor Ann Gray all contributed to this article.

Virginia has not had a case of human rabies since 1953, but epidemiologists at the Virginia Department of Health fear that exemplary record is at risk from an outbreak of rabies among wild animals.[1] In 1982, Virginia was second only to Texas in the number of rabid wild animals reported over the nation. The score: Texas 785, Virginia 745.

The infection in Virginia is focused in raccoons. Of those 745 rabid wild animals reported last year, 645 were raccoons. The record thus far for 1983, as this was written, showed 249 cases of rabies in racccoons out of a total of 274 reported cases of wild animal rabies. The infection has spread very fast; in 1980 there were only seven reported cases of raccoon rabies in Virginia.

Locus of the outbreak has been Virginia's northern counties. Three-fourths of the raccoon rabies incidence in 1982 was reported in Fairfax and Loudoun counties; Prince William and Fauquier counties also registered increased incidence. In recent months, however, the disease has spread southward and eastward, with cases newly reported in Greene, Highland, Orange, Stafford and Westmoreland counties.

Why the rabies outbreak among raccoons? Epidemiologists do not know.[2] Rabies breaks out from time to time in wild animals all over the world. Raccoons are the major rabies reservoir in Alabama, Florida, Georgia and South Carolina, but historically in Virginia the disease has primarily affected foxes; it is speculated that hunters imported infected raccoons from the southeast into the Virginia area. Now the outbreak has spread into the District of Columbia, Maryland and Pennsylvania, and in Delaware the first rabid raccoon was recently reported.

In an effort to contain the dis-

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RABIES POST-EXPOSURE PROPHYLAXIS GUIDE

These recommendations from the Virginia Department of Health are only a guide. In applying them, take into account the animal species involved, circumstances of the bite or other exposure, vaccination status of the animal, and presence of rabies in the region. Local or state public health officials should be consulted if questions arise.

All bites and wounds should immediately be cleansed thoroughly with soap and water. If anti-rabies treatment is indicated, both rabies immune globulin (RIG) and human diploid cell rabies vaccnine (HDCV) should be given as soon as possible, regardless of the interval of exposure.

Domestic animals: dog, cat

- If, at the time of the attack, the animal is healthy and available for ten days of observation, do not treat the exposed person unless rabies develops in the animal. At the first sign of rabies in the animal, begin treatment with RIG and HDCV. The symptomatic animal should be killed immediately and tested.
- If, at the time of the attack, the animal is rabid or suspected of being rabid, administer RIG and HDCV. If RIG is not available, use anti-rabies serum, equine (ARS), at no more than the recommended dosage.
- If the attacking animal has escaped or is otherwise unknown, consult public health officials. If treatment is indicated, give RIG and HDCV.

Note: If HDCV is not available, use duck embryo vaccine (DEV). Local reactions to vaccines are common and do not contraindicate continuing treatment. Discontinue vaccine if fluorescent antibody tests of the animal are negative.

Wild animals: raccoon, fox, skunk, bat, coyote, bobcat and other carnivores

- The attacking animal should be considered rabid unless proven negative by laboratory tests. Holding the animal for observation is not recommended; the animal should be killed and tested as soon as possible. Treat the exposed person with RIG and HDCV.

Other animals: livestock, rodents, rabbits and hares

- Consider these cases individually and consult with public health officials about the need for prophylaxis. Bites of squirrels, hamsters, guinea pigs, gerbils chipmunks, rats, mice, other rodents, rabbits and hares almost never call for anti-rabies prophylaxis.

VSBUREAU

ease, Dr. James B. Kenley, State Commissioner of Health, recently asked the Commission of Game and Inland Fisheries to institute a year-round ban on intrastate relocations of raccoons. He noted that the Tidewater area is a primary source for trapping raccoons for translocation to Southwest Virginia, where the raccoon population is quite sparse.

The outbreak places humans at risk not only through contact with rabid wild creatures but through domestic pets that may come in contact with rabid wild animals. Ten cases of rabies in cats were reported last year, and in Frederick County early this year the owners of a cat were given rabies vaccine when the animal was found to be rabid. There is a new vaccine for cats, protective for three years.

On Dr. Kenley's recommendation, five counties in Northern Virginia—Loudoun, Fairfax, Arlington, Prince William and Frederick—have passed local ordinances requiring the vacination of cats against rabies.

More than 250 Virginians were treated last year alone for exposure to proven or suspected rabid animals. Fortunately, a new rabies vaccine made their post-exposure therapy both less dangerous and less painful than previously.

The human diploid cell rabies vaccine (HDCV), approved by the Federal Drug Administration in 1980, is a tremendous improvement over the duck embryo vaccine (DEV) that had been in use since 1958 and produced such a high rate of complications. Studies showed that serious systemic problems developed in 10% of patients given DEV, including anaphylaxis, transverse myelitis and meningioencephalitis.[3] Moreover, after the standard vaccination regimen a satisfactory antibody level was achieved in only 66% of patients.[4]

The new vaccine is a different story. Complications of anaphylaxis or neurologic dysfunction have been reported rarely with HDCV. Local reactions, such as pain and redness in the site of injection, have occurred in 25% of cases, and systemic reactions, including dizziness, headaches, malaise, shortness of breath and fever, have occurred in about 4%. There appear to have been no deaths resulting from its use.[5] Admittedly, it is a new drug and other complications may occur with greater use.

The human diploid cell vaccine has achieved protective antibody levels in 99% of the persons who received it, a substantial improvement over the duck embryo vaccine.[6]

Editors' Note: The even newer rhesus diploid cell strain rabies vaccine (RDRV) has shown promising results in clinical trials (JAMA 249: 2663–2665, 1983).

First distributed only through the Department of Health, HDCV is now also available at hospital pharmacies. The cost is about $45 per cc. The vaccine is used for both pre- and post-exposure treatment.

Pre-exposure prophylaxis is recommended for certain high risk groups such as veterinarians, animal handlers, trappers, some laboratory workers, and persons who remove the heads of animals suspected of being rabid.

The pre-exposure dosage of HDCV recommended by the Food and Drug Administration is three 1-ml doses, one each on days 0, 7, and either 21 or 28. A different regimen has been recommended, after examination of data, by the Advisory Committee on Immunization Practices at the Centers for disease Control: three 0.1-ml doses, one each on days 0, 7, and either 21 or 28, given intradermally. Approval of the committee's schedule by the Federal Drug Administration awaits data from the vaccine manufacturer demonstrating the effectiveness of the smaller regimen and a change in packaging to reflect the smaller dose.

As for post-exposure prophylaxis, the recommended HDCV schedule is five 1-ml doses, one each on days 0, 3, 7, 14 and 28, given intramuscularly. Rabies immune globulin (RIG) should always be given in conjunction with post-exposure therapy on the same day as the first dose of the vaccine, the RIG dosage to be calculated according to body weight. The administration of rabies immune globulin confers passive immunity until the HDCV stimulates antibody production; the only failures with the human diploid cell vaccine have occurred when RIG was not administered simultaneously in post-exposure therapy. (*cont'd over*)

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Council considers reports, recommendations from spheres of Society activity

With Dr. Harold L. Williams presiding, the Medical Society of Virginia's Council met at MSV headquarters in Richmond on April 16 to consider aspects of the Society's activity, including the following.

• From Mrs. Gerald Weitzman, president of The Medical Society of Virginia **Auxiliary**, came the report that AMA-ERF contributions for 1983 were already in excess of $36,000.

The Auxiliary has launched a new state-wide project, she continued, an "Organ Donor Awareness Program," in which auxilians will be urged to fill out the organ donor documents on their driver's licenses. The program is part of a national effort, with its own officially designated week, and brought to the Auxiliary a citation from Virginia's Governor Robb.

• Reporting on the Society's **insurance program**, Dr. Alvin E. Conner, chairman of the Insurance Committee, said that the professional liability insurance experience for the year 1981–1982 "looked good," but figures for the 1982–1983 term thus far were "not encouraging." Hoped-for dividends under the new retrospective plan might be forthcoming later this year, he projected. The plan is carried by St. Paul Fire & Marine.

As for the Society's Blue Cross Blue Shield medical insurance plan, the first quarter's experience this year held the cost line nicely, Dr. Conner said. There have been suggestions, he observed, that this medical insurance plan be put out for bids from all carriers, in which connection he reminded the councilors that a distinct advantage in the Blue Cross Blue Shield alliance is the firm's willingness to write a policy for anyone who wants it, whereas other firms have underwriting requirements that screen out some applicants.

The plans offered through the David A. Dyer firm are doing "exceptionally well," Dr. Conner concluded.

The Council commended Dr. Conners and the members of the Insurance Committee for their long hours in treating successfully the rash of problems of the past few years.

• Hot topics were promised by Dr. Kinloch Nelson for the **scientific program** at the Society's annual meeting in November at the Homestead. Dr. Nelson is Director of Continuing Education.

Because of the new vaccine's high level of immunologic response, post-vaccination titers to confirm immunity are not recommended, except in the case of immunocompromised patients or patients who are in continuous contact with rabies virus, such as laboratory workers.

What are the indications for administration of the vaccine? See the accompanying Guide, written from information provided by the Department of Health's Epidemiology Division.[5]

1. Rabies erupts in Virginia. Va Med New Bureau 109:355, 1982
2. MMWR 31:592-593, 1982
3. Plotkin SA: Rabies vaccination in the 1980s. Hosp Prac 15:65-72, 1980
4. Burridge MJ, Baer GM, Summer JW: Intradermal immunization with human diploid cell rabies vaccine. JAMA 248:1611-1614, 1982
5. Rabies Prevention. MMWR 29:265-284, 1980
6. Supplementary Statement on Rabies Vaccine and Serologic Testing. MMWR 30:535, 1981

Hotel rate benefit at MSV meeting

When summer's here, The Medical Society of Virginia's annual meeting cannot be far away. This year's dates: November 3–6. The setting: the Homestead in Hot Springs. You will find rate information with the reservation blank on page 339 of this issue.

For those attending the meeting, Homestead officials have set up a rate differential that applies to the hotel bill, like this: For double occupancy @ $95, the amount of $71.25 is designated as the expense of the meeting participant, and $23.75 as the expense of the second occupant. The differential applies similarly to the parlor rates.

For the participant's records, the differential will be declared on the statement issued when the bill is paid.

Computers will be the subject on Friday afternoon, November 5, he announced, their use first in continuing medical education, such as the AMA's Telenet system, and second as educational tools to help the physician test the effectiveness of patient workup and treatment and evaluate costs.

On Saturday morning November 6 the program will focus on preferred provider organizations and prospective hospital reimbursement by diagnostic-related group, or DRGs.

• The recent AMA **Leadership** Conference in Chicago drew 800 officers and staff members of state and county societies, reported Dr. C. Barrie Cook, MSV President Elect, who was himself among those at the conference as the representative of President Williams.

• The councilors approved publication in 1984 of a second edition of the Medical Society of Virginia **Membership Directory** that comprised the September 1982 issue of VIRGINIA MEDICAL. Well in advance, promotional pages in the journal and mailings to members will remind members to notify of changes of address.

• The course of medically oriented **legislation** at the 1983 General Assembly generated a briefing by Allen C. Goolsby III, the Society's counsel. See pages 368-372 of this issue for a review of that action.

• Three recommendations brought by Dr. Ira J. Green on behalf of the **Vanguard** Committee met the Council's approval: 1) that the Vanguard Committee continue to monitor federal legislation pertaining to quality medical care and alert the membership to its findings; 2) that the committee evaluate the Department of Health and Human Services' proposed Health Care Incentive Program, which relates primarily to catastrophic illness, cost-sharing, and reimbursement methods and rates, and make recommendations to the Council; and 3) that the committee should continue to monitor federal block grants.

• A survey of the 1200 physicians performing their **residencies** in Virginia is to be undertaken by the Membership Committee, related Dr. J. Thomas Hulvey, chairman of the committee. The purpose: to plumb sentiment on the proposed residents section for The Medical Society of Virginia's House of Delegates.

• An all-day workshop to train intervenors and investigators for the **Physicians Health and Effectiveness** Committee was forecast for early May by the committee's chairman, Dr. William H. Barney.

The Council will next meet on September 10, again at the Society's Richmond headquarters.

VIRGINIA MEDICAL

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Brief Summary of prescribing information

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TABLETS

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CONTRAINDICATIONS: Hypersensitivity to antihistamines or sympathomimetics. Ru-Tuss Tablets are contraindicated in children under 12 years of age and in patients with glaucoma, bronchial asthma and women who are pregnant. Concomitant use of MAO inhibitors is contraindicated.

WARNINGS: Ru-Tuss Tablets may cause drowsiness. Patients should be warned of possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers.

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OVERDOSAGE: Since the action of sustained release products may continue for as long as 12 hours, treatment of overdoses directed at reversing the effects of the drug and supporting the patient should be maintained for at least that length of time. Saline cathartics are useful for hastening evacuation of unreleased medication. In children and infants, antihistamine overdosage may produce convulsions and death.

ADVERSE REACTIONS: Hypersensitivity reactions such as rash, urticaria, leukopenia agranulocytosis, and thrombocytopenia may occur. Other adverse reactions to Ru-Tuss Tablets may be drowsiness, lassitude, giddiness, dryness of the mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, dizziness and insomnia. Large overdoses may cause tachypnea, delirium, fever, stupor, coma and respiratory failure.

DOSAGE AND ADMINISTRATION: Adults and children over 12 years of age, one tablet morning and evening. Not recommended for children under 12 years of age. Tablets are to be swallowed whole.

Federal law prohibits dispensing without prescription.

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Rx ONLY **Berocca® Plus** TABLETS

THE MULTIVITAMIN/MINERAL FORMULATION

Before prescribing, please consult complete product information, a summary of which follows:

Each Berocca® Plus tablet contains 5000 IU vitamin A (as vitamin A acetate), 30 IU vitamin E (as *dl*-alpha tocopheryl acetate), 500 mg vitamin C (ascorbic acid), 20 mg vitamin B_1 (as thiamine mononitrate), 20 mg vitamin B_2 (riboflavin), 100 mg niacin (as niacinamide), 25 mg vitamin B_6 (as pyridoxine HCl), 0.15 mg biotin, 25 mg pantothenic acid (as calcium pantothenate), 0.8 mg folic acid, 50 mcg vitamin B_{12} (cyanocobalamin), 27 mg iron (as ferrous fumarate), 0.1 mg chromium (as chromium nitrate), 50 mg magnesium (as magnesium oxide), 5 mg manganese (as manganese dioxide), 3 mg copper (as cupric oxide), 22.5 mg zinc (as zinc oxide)

INDICATIONS: Prophylactic or therapeutic nutritional supplementation in physiologically stressful conditions, including conditions causing depletion, or reduced absorption or bioavailability of essential vitamins and minerals, certain conditions resulting from severe B-vitamin or ascorbic acid deficiency, or conditions resulting in increased needs for essential vitamins and minerals

CONTRAINDICATIONS: Hypersensitivity to any component

WARNINGS: Not for pernicious anemia or other megaloblastic anemias where vitamin B_{12} is deficient. Neurologic involvement may develop or progress, despite temporary remission of anemia, in patients with vitamin B_{12} deficiency who receive supplemental folic acid and who are inadequately treated with B_{12}.

PRECAUTIONS: *General*: Certain conditions may require additional nutritional supplementation. During pregnancy, supplementation with vitamin D and calcium may be required. Not intended for treatment of severe specific deficiencies. *Information for the Patient*: Toxic reactions have been reported with injudicious use of certain vitamins and minerals. Urge patients to follow specific dosage instructions. Keep out of reach of children. *Drug and Treatment Interactions*: As little as 5 mg pyridoxine daily can decrease the efficacy of levodopa in the treatment of parkinsonism. Not recommended for patients undergoing such therapy.

ADVERSE REACTIONS: Adverse reactions have been reported with specific vitamins and minerals, but generally at levels substantially higher than those in Berocca Plus. However, allergic and idiosyncratic reactions are possible at lower levels. Iron, even at the usual recommended levels, has been associated with gastrointestinal intolerance in some patients.

DOSAGE AND ADMINISTRATION: Usual adult dosage: one tablet daily. Not recommended for children. Available on prescription only.

HOW SUPPLIED: Golden yellow, capsule-shaped tablets — bottles of 100

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Candidates for nutritional therapy...

5,000,000 hospital patients with infections.[1] Many are anorectic and may have a markedly reduced food intake. Supplements are often provided as a prudent measure because the vitamin status of critically ill patients cannot be readily determined.[2]

Berocca Plus. A balanced formula for prophylactic or therapeutic nutritional supplementation. Berocca Plus Tablets provide: therapeutic levels of ascorbic acid and B-complex vitamins; supplemental levels of biotin, vitamins A and E, and five important minerals (iron, chromium, manganese, copper and zinc); plus magnesium. Berocca Plus is not intended for the treatment of specific vitamin and/or mineral deficiencies.

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THE MULTIVITAMIN/MINERAL FORMULATION

1. Dixon RE: *Ann Intern Med* 89(Part 2):749-753, Nov 1978. 2. Shils ME, Randall HT: Diet and nutrition in the care of the surgical patient, chap. 36, in *Modern Nutrition in Health and Disease*, edited by Goodhart RS, Shils ME; Philadelphia, Lea & Febiger, 1980, p. 1114

Please see summary of product information on reverse page.

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LETTERS

Successful "lock-in" program expanded to control Medicaid abuse

Effective March 1, 1983, the Virginia Department of Health expanded its Medicaid "lock-in" program. The term lock-in refers to Medicaid utilization control. A lock-in program of limited scope has been supported by Virginia physicians and pharmacists, but now a new lock-in plan has been designed to reach a larger patient population and to promote the proper medical management of essential health care, while reducing costs associated with unnecessary services. We cannot continue to pay when a recipient goes to several physicians for the same service or has multiple prescriptions filled for the same drug.

Recipients found to be high utilizers of unnecessary Medicaid services, particularly through emergency rooms, outpatient office visits and prescription drugs, are identified as candidates for the lock-in program. These recipients are notified that they must select a mutually agreeable primary care physician and designated pharmacy. The primary care physician provides the necessary routine medical care and makes medically necessary referrals to specialists. In the absence of the primary physician, a physician covering for him may provide routine care as needed. The designated pharmacy is to supply all the recipient's prescription drugs. Provisions are made to ensure that recipients may receive services from any participating provider in *true* emergency situations. A lock-in recipient will have the names and provider numbers of his primary care physician and designated pharmacy printed on the face of his eligibility card.

Less than 8,000 of about 368,000 Medicaid recipients are expected to be maintained on lock-in at any one time. Recipients' utilization patterns are reviewed at least every 12 months to determine whether or not they should remain in the program. The recipient whose utilization pattern returns to normal levels may withdraw from the program. However, it may be in the recipient's best interest, especially the legitimate high-utilizer with multiple medical problems, to remain on the program.

Comments regarding the lock-in program have

generally been favorable. Most recipients have been receptive once they understand the program's purpose and are assured that they will receive the services necessary to meet their medical needs.

Lock-in is not a panacea for all Medicaid utilization abuse, but is a positive step forward and is simple, with a minimum of bureaucracy. Most importantly, it has been accomplished through the cooperation of Virginia physicians and pharmacists.

James B. Kenley, MD,
State Commissioner of Health

109 Governor Street
Richmond VA 23219

Urges linguistic caution in a litigious climate

In the brochure on breast cancer treatment that appeared in the November issue of VIRGINIA MEDICAL, it seems to me that the first sentence on the last page could be misleading.[1] Under the subhead of "Your Recovery from Breast Cancer," the text begins, "Breast cancer is a curable disease, so your emotional health is also an important factor in recovery."

Medically oriented people know that cancer is curable in a certain percentage of patients, but those who have cancer could interpret "a curable disease" to mean a total expectancy of cure. It seems to me it would have been appropriate to say that breast cancer is a curable disease in the majority of patients, or a percentage of patients, whichever is suitable.

From a medico-legal point of view alone, accuracy of information is highly desirable these days.

Richard H. Fisher, MD
Lewis-Gale Clinic
1802 Braeburn Drive, Salem VA 24153

1. Breast Cancer Treatments: a Helpful Guide (brochure). Va Med 110:757, 1983

Editors' Note: The brochure to which Dr. Fisher refers was developed by a committee of which Dr. Gerald Goldstein, Charlottesville, was chairman. Dr. Goldstein replies as follows:

The comments of Dr. Fisher are welcomed and appreciated. I agree, and think that it would be better to state ". . . . breast cancer is often curable . . ." The committee that prepared this brochure is eager to receive criticism or suggestions to enable its members to improve this brochure. Any correspondence should be sent to Virginia Medical or to the American Cancer Society, Virginia Division, PO Box 7288, Richmond VA 23221.

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MEDICINE AND THE LAW

Medical Records: Their Care and Keeping

The following information on medical records was provided by Allen C. Goolsby III, counsel for The Medical Society of Virginia.

Ownership

The physician owns the medical records. He has the responsibility of protecting them from damage and unauthorized access. The patient has the right, however, to request copies of the record.

When a physician works for a professional corporation, there often is uncertainty as to whether the medical records belong to the corporation or the individual physician. The answer will depend on the facts of each particular case. It is recommended that this issue be resolved and confirmed in writing by the participants when the relationship is established. If problems develop among the physicians in the corporation, the dispute is much easier to settle if the ownership of records issue already has been resolved.

Contents

Generally, medical records should contain: 1) identification data; 2) medical history of the patient; 3) reports of physical examinations; 4) diagnostic and therapeutic orders; 5) evidence of appropriate informed consent; 6) clinical observations; 7) reports of procedures, tests and their results; 8) conclusions at the termination of treatment and evaluation.

Maintenance

Medical records may be maintained in a written form, on microfilm or on a computer. The use of microfilm has been specifically recognized in the Virginia Code, which provides that microphotographs of medical records are admissible as evidence any time the originals would be admissible. While there is no specific recognition of computerized medical records, neither is there any specific prohibition. When the records are computerized or microfilmed, care must be taken to protect the confidentiality of the records and restrict access to them.

Retention

Records should be retained for at least as long as they would be needed to defend the physician in a law suit. In the case of personal injury actions, that period is two years from the date of injury. A suit on an unwritten or implied contract must be brought within three years. If the patient is a minor or an incompetent, the statute of limitations is suspended until the minor reaches the age of 18 or until the incompetent becomes competent. For these patients, records should be retained for at least three years after those dates.

Medicare regulations require that records relating to Medicare health insurance claims be retained for five years after the cost report on that patient has been filed. The Occupational Safety and Health Administration requires that records of patients who are seen by a physician under a contract with a company be held for the term of the employee's employment plus 30 years.

The American Medical Records Association recommends holding all records for ten years after the most recent patient care, and the Richmond Academy of Medicine suggests a seven-year holding period.

The physician should consider all these recommendations in the light of the characteristics of his practice.

Access

The Code of Virginia requires a physician to furnish copies of the records to the patient or his attorney within 15 days after a written request has been made. The physician may charge the patient a reasonable fee. If the physician refuses to provide the copies, the patient or his attorney may subpoena the records and, if the refusal was willful, the court may award damages to the patient for all expenses incurred to obtain the copies.

If the physician includes in the record a written statement that, in his opinion, furnishing the patient with a copy of his record would be injurious to the patient's health or well-being, then the Virginia Code allows for the records to be released only to

the patient's attorney. The statute also provides to whom records of a deceased or incompetent patient must be furnished.

Although there is no provision concerning a minor's medical records, they generally can be released to a parent or guardian with the minor's consent unless the minor 1) is or has been married; 2) received treatment for mental illness or substance abuse; 3) received medical services for birth control, pregnancy or family planning or for testing or treatment of venereal or other reportable contagious disease.

Other exceptions to confidentiality include the Virginia Code's requirement that Virginia physicians report child abuse, blindness, deafness, cancer, wounds inflicted by weapons, and the diseases specified as reportable by the Virginia State Board of Health and as shown on this page.

Also, physicians must 1) furnish copies of an injured employee's record for workmen's compensation purposes to the employee, employer, and insurance carrier; 2) disclose to the payor information from the records of a mental health patient who has requested third-party payment of a physician's bill; 3) follow OSHA regulations when providing access to the records of patients protected by occupational health laws.

The physician who is required to disclose records or papers should include copies of X-rays, health care records, test records, prescriptions and other information used to assess the patient's health condition. Virginia courts may also require a physician's personal notes to be included, especially if they are maintained with the patient's record.

Disposal

Generally, records may be disposed of by shredding or burning. Again, the primary concern is to maintain the confidentiality of the record. It has been suggested that destruction be certified and that the certificate kept permanently, but there is no legal requirement for this.

CLIP AND KEEP FOR REFERENCE

In Virginia, these diseases must be reported

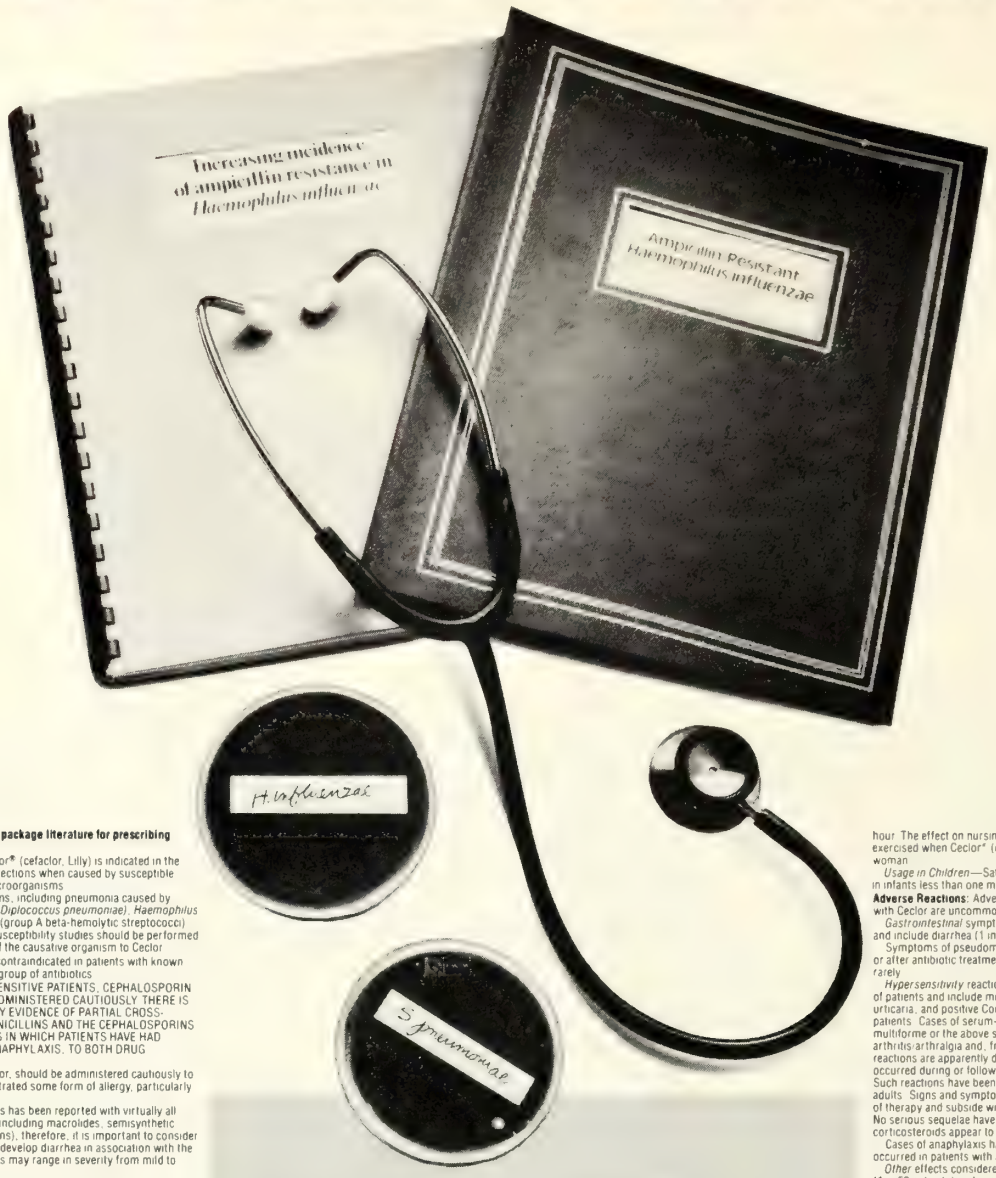
Amebiasis
***Anthrax**
Arboviral infections
Aseptic meningitis
Bacterial meningitis
***Botulism**
Brucellosis
Campylobacter infections
Chancroid
Chickenpox
Congenital rubella syndrome
***Diphtheria**
Encephalitis, primary and post-infectious
***Foodborne diseases**
Gonorrhea
Granuloma inguinale
Hepatitis A (infectious); B (serum); and unspecified
Histoplasmosis
Influenza (number of cases only)
Kawasaki's disease
Legionellosis
Leprosy
Leptospirosis
Lymphogranuloma venereum
Malaria
***Measles (rubeola)**
Meningococcal infections
Mumps
***Nosocomial outbreaks**
Occupational illnesses
Ophthalmia neonatorum
Pertussis
Phenylketonuria (PKU)
***Plague**
***Poliomyelitis**
***Psittacosis**
Q fever
***Rabies in man**
Post-exposure rabies treatment
Rabies in animals
Reye's syndrome
Rocky Mountain spotted fever
Rubella
Salmonellosis
Shigellosis
***Smallpox**
***Syphilis**
Tetanus
****Toxic substance related illness**
Trichinosis
Tularemia
***Tuberculosis**
Typhoid fever
Typhus, flea-borne
Vibrio infections, including cholera*
Waterborne outbreaks
***Yellow fever**

*Because of their extremely contagious nature or potential for harm, these diseases are to be reported with all speed by telephone or other telecommunication.

**Illnesses or diseases resulting from exposure to toxic substance include, but are not limited to, occupational lung disease (silicosis, asbestosis, byssinosis) and occupationally-related cancers (mesothelioma).

Report incidence or suspicion of these diseases to the Health Department in your locality, either by telephone or on one of the forms provided by Virginia's Division of Epidemiology. For a supply of those forms, write or call Grayson B. Miller, Jr., MD, State Epidemiologist, 109 Governor St., Richmond VA 23219, (804) 786-6261.

An added complication... in the treatment of bacterial bronchitis*


Increasing incidence of ampicillin resistance in Haemophilus influenzae
Ampicillin Resistant Haemophilus influenzae
H. influenzae
S. pneumoniae

Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor

Pulvules®, 250 and 500 mg

Brief Summary. Consult the package literature for prescribing information

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions: *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor® (cefaclor, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below.

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

1061782R1

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.*

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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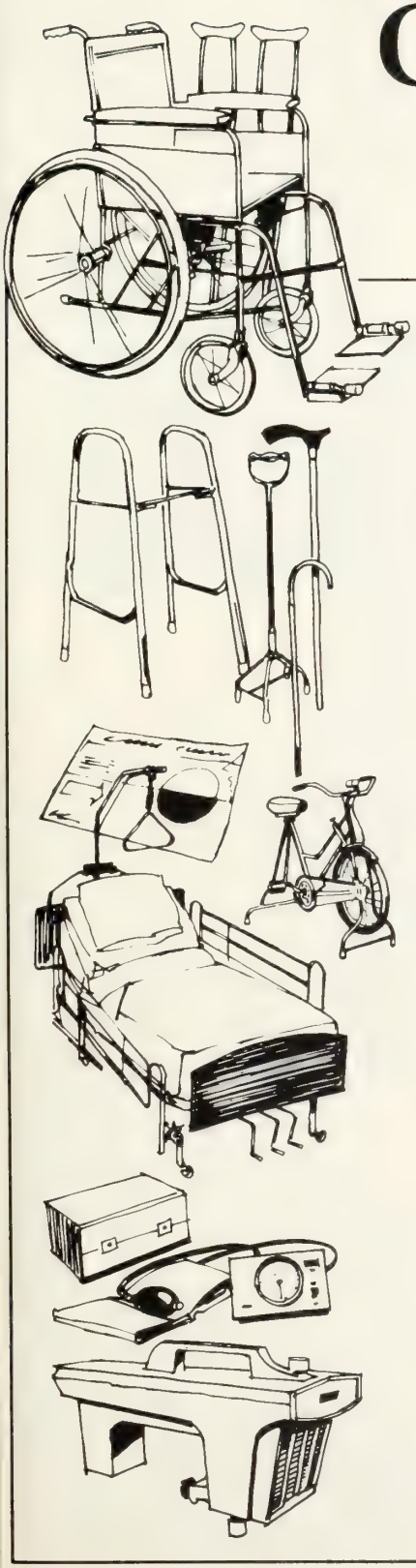

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Saint Albans Psychiatric Hospital

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Pain—triggering anxiety—which accentuates the perception of pain...together they're worse than either alone. And since they're usually both present in musculoskeletal disorders, the best therapy is often a combination of analgesic and anxiolytic agents. Equagesic®-M combines the pain relief of aspirin with the tension-reducing properties of meprobamate—because together they're better than either alone.

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Proven superior to aspirin alone in controlled clinical trials

(BRIEF SUMMARY)

DESCRIPTION: Each tablet contains 200 mg meprobamate and 325 mg aspirin.

INDICATIONS Adjunct in short-term treatment of pain accompanied by tension and/or anxiety in patients with musculoskeletal disease. Clinical trials demonstrated in these situations relief of pain is somewhat greater than with aspirin alone. Effectiveness in long-term use, i.e. over 4 months, has not been assessed by systematic clinical studies. Physicians should periodically reassess usefulness of drug for individual patients.

CONTRAINDICATIONS. ASPIRIN: Allergic or idiosyncratic reactions to aspirin or related compounds. MEPROBAMATE: Acute intermittent porphyria, allergic or idiosyncratic reactions to meprobamate or related compounds, e.g. carisoprodol, mebutamate, or carbromal.

WARNINGS ASPIRIN: Use salicylates with extreme caution in patients with peptic ulcer, asthma, coagulation abnormalities, hypoprothrombinemia, vitamin K deficiency, or those on anticoagulants. In rare instances, aspirin in persons allergic to salicylates may result in life threatening allergic episodes.

MEPROBAMATE: DRUG DEPENDENCE: Physical and psychological dependence, and abuse have occurred. Chronic intoxication from prolonged ingestion of, usually, greater than recommended doses is manifested by ataxia, slurred speech, and vertigo. Therefore, carefully supervise dose and amounts prescribed and avoid prolonged use, especially in alcoholics and others with known propensity for taking excessive quantities of drugs. Sudden withdrawal after prolonged and excessive use may precipitate recurrence of preexisting symptoms, e.g. anxiety, anorexia, or insomnia, or withdrawal reactions, e.g., vomiting, ataxia, tremors, muscle twitching, confusional states, hallucinosis, and, rarely, convulsive seizures. Such seizures are more likely in persons with CNS damage or preexistent or latent convulsive disorders. Onset of withdrawal symptoms occurs usually within 12 to 48 hours after discontinuation, symptoms usually cease within next 12-to-48-hour period. When excessive dosage has continued for weeks or months, reduce dosage gradually over 1 to 2 weeks rather than stop abruptly. Alternatively, a short-acting barbiturate may be substituted, then gradually withdrawn.

POTENTIALLY HAZARDOUS TASKS: Warn patients meprobamate may impair mental or physical abilities required for potentially hazardous tasks, e.g., driving or operating machinery.

ADDITIVE EFFECTS: Since CNS-suppressant effects of meprobamate and alcohol or meprobamate and other psychotropic drugs may be additive, exercise caution with patients taking more than one of these agents simultaneously.

USAGE IN PREGNANCY AND LACTATION. An increased risk of congenital malformations associated with minor tranquilizers (meprobamate, chlordiazepoxide, and diazepam) during first trimester of pregnancy, has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at time of institution of therapy should be considered. Advise patients if they become pregnant during therapy or intend to become pregnant to communicate with their physicians about desirability of discontinuing the drug.

Meprobamate passes the placental barrier. It is present both in umbilical-cord blood at or near maternal plasma levels and in breast milk of lactating mothers at concentrations two to four times that of maternal plasma. When use of meprobamate is contemplated in breastfeeding patients, consider the drug's higher concentrations in breast milk as compared to maternal plasma levels.

USAGE IN CHILDREN: Keep preparations with aspirin out of reach of children. Equagesic®-M is not recommended for patients 12 years of age and under.

PRECAUTIONS ASPIRIN: Salicylates antagonize uricosuric activity of probenecid and sulfinpyrazone. Salicylates are reported to enhance hypoglycemic effect of sulfonylurea antidiabetics.

MEPROBAMATE: Use lowest effective dose, particularly in elderly and/or debilitated, to preclude over-sedation. Meprobamate is metabolized in the liver and excreted by the kidney, to avoid excess accumulation exercise caution in its use in patients with compromised liver or kidney function. Meprobamate occasionally may precipitate seizures in epileptic patients. It should be prescribed cautiously and in small quantities to patients with suicidal tendencies.

ADVERSE REACTIONS. ASPIRIN: May cause epigastric discomfort, nausea, and vomiting. Hypersensitivity reactions, including urticaria, angioneurotic edema, purpura, asthma, and anaphylaxis may rarely occur. Patients receiving large doses of salicylates may develop tinnitus.

MEPROBAMATE: CNS: Drowsiness, ataxia, dizziness, slurred speech, headache, vertigo, weakness, paresthesias, impairment of visual accommodation, euphoria, overstimulation, paradoxical excitement, fast EEG activity.

GI: Nausea, vomiting, diarrhea.

CARDIOVASCULAR: Palpitation, tachycardia, various forms of arrhythmia, transient ECG changes, syncope, hypotensive crisis.

ALLERGIC OR IDIOSYNCRATIC: Milder reactions are characterized by itchy, urticarial, or erythematous maculopapular rash, generalized or confined to the groin. Other reactions include leukopenia, acute nonthrombocytopenic purpura, petechiae, ecchymoses, eosinophilia, peripheral edema, adenopathy, fever, fixed drug eruption with cross-reaction to carisoprodol, and cross-sensitivity between meprobamate, mebutamate and meprobamate-carbromal. Rare, more severe hypersensitivity reactions include hyperpyrexia, chills, angioneurotic edema, bronchospasm, oliguria, and anuria. Also, anaphylaxis, exfoliative dermatitis, stomatitis, and proctitis. Stevens-Johnson syndrome and bullous dermatitis have occurred.

HEMATOLOGIC (SEE ALSO "ALLERGIC OR IDIOSYNCRATIC"): Agranulocytosis, aplastic anemia have been reported, although no causal relationship has been established, and thrombocytopenic purpura.

OTHER: Exacerbation of porphyric symptoms.

DOSAGE AND ADMINISTRATION. Usual dose is one or two tablets, 3 to 4 times daily as needed for relief of pain when tension or anxiety is present. Not recommended for patients 12 years of age and under.

OVERDOSAGE: Treatment is essentially symptomatic and supportive. Any drug remaining in the stomach should be removed. Induction of vomiting or gastric lavage may be indicated. Activated charcoal may reduce absorption of both aspirin and meprobamate. Aspirin overdosage produces usual symptoms and signs of salicylate intoxication. Observation and treatment should include management of hyperthermia, specific parenteral electrolyte therapy for ketoacidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole-blood transfusions. Suicidal attempts with meprobamate have resulted in drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse. Some suicidal attempts have been fatal. The following data, reported in the literature and from other sources, are not expected to correlate with each case (considering factors such as individual susceptibility and length of time from ingestion to treatment), but represent usual ranges reported. Acute simple overdose (meprobamate alone): Death has been reported with ingestion of as little as 12 gram meprobamate and survival with as much as 40 gram.

BLOOD LEVELS

0.5-2.0 mg percent represents usual blood-level range after therapeutic doses. The level may occasionally be as high as 3.0 mg percent.

3-10 mg percent usually corresponds to findings of mild-to-moderate symptoms of overdosage, such as stupor or light coma.

10-20 mg percent usually corresponds to deeper coma, requiring more intensive treatment. Some fatalities occur.

At levels greater than 20 mg percent, more fatalities than survivals can be expected.

Acute combined overdose (meprobamate with other psychotropic drugs or alcohol): Since effects can be additive history of ingestion of a low dose of meprobamate plus any of these compounds (or of a relatively low blood or tissue level) cannot be used as a prognostic indicator.

In cases of excessive doses, sleep ensues rapidly and blood pressure, pulse, and respiratory rates are reduced to basal levels. Any drug remaining in stomach should be removed and symptomatic treatment given. Should respiration or blood pressure become compromised, respiratory assistance, CNS stimulants, and pressor agents should be administered cautiously as indicated. Diuresis, osmotic (mannitol) diuresis, peritoneal dialysis, and hemodialysis have been used successfully in removing both aspirin and meprobamate. Alkalinization of the urine increases excretion of salicylates. Careful monitoring of urinary output is necessary, and caution should be taken to avoid overhydration.

Relapse and death, after initial recovery have been attributed to incomplete gastric emptying and delayed absorption.

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Hospitality House
Williamsburg, Va

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☐ Afternoon Session-12:30 p.m.-4:30 p.m.
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MEETINGS ABOUT MEDICINE

FREE FILM SHOWS PHYSICIAN ON TRIAL

A video tape of a mock malpractice trial is being offered free of charge by the Education Committee of The Medical Society of Virginia for showing at meetings of physicians. The film illustrates what a practicing physician is up against when he or she is sued for malpractice and the case goes to court. It is 45 minutes long and shows the four key elements in a medical malpractice trial: testimony and cross-examination of the plaintiff, the expert witness and the defendant, and the judge's instructions to the jury.

Developed by the Visual Education Department of the Medical College of Virginia/Virginia Commonwealth University, the film is sponsored by The Medical Society of Virginia and the St. Paul Fire & Marine Insurance Companies.

If you wish to borrow the film, call or write Kinloch Nelson, MD, CME Coordinator, at MSV headquarters, 4205 Dover Road, Richmond VA 23221, (804) 353-2721.

June 23-30
Sports and Tennis Medicine Symposium (Georgetown University/Virginia Sports Medicine and Rehabilitation Institute), *Wimbledon, England*. Cat. I. Robert P. Nirschl, MD, 3801 N. Fairfax Dr., Arlington VA 22203, (703) 525-8600.

June 24-26
Internal Medicine Symposium (Eastern Virginia Medical School), *Virginia Beach*. James P. Baker, MD, EVMS, PO Box 1980, Norfolk VA 23510.

July 1-3
13th Annual Sports Medicine Symposium (North Carolina Medical Society), Blockade Runner, *Wrightsville, North Carolina*. Fee: $20. Alan Skipper, North Carolina Medical Society, PO Box 27167, Raleigh NC 27611, (919)833-3836.

July 15-17
Ambulatory Electrocardiography (International Medical Education Corporation), *Virginia Beach*. 13 credit hrs. Fee, $260. IMEC, 64 Inverness Dr., E, Englewood CO 80112, toll free, (800) 525-8651.

July 22-24
Advances in Cardiology (Medical College of Virginia), *Hot Springs*. 12 credit hrs. Fee, $230. Randy Casey, Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

July 22-24
Annual Golf Classic Symposium: Selected Topics for the Primary Care Physician (Eastern Virginia Medical School), *Williamsburg*. Robert L. Cassidy, MD, EVMS, PO Box 1980, Norfolk VA 23501.

June 25-26
Diagnostic Ultrasound (University of Virginia), *Charlottesville*. 5 credit hrs. Fee: $45. Charles D. Teates, MD, Box 486, University of Virginia Medical Center, Charlottesville VA 22908, (804) 924-5201.

July 28-30
5th Annual Pediatric Primary Care Conference (Medical College of Virginia), *Virginia Beach*. 12¾ Cat. I credits. Fee: $250. Sheri Rosner, Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

July 28-30
Internal Medicine Summer Seminar (Eastern Virginia Medical School), *Virginia Beach*. James P. Baker, MD, EVMS, PO Box 1980, Norfolk VA 23510.

August 1-6
Sports and Tennis Medicine Symposium (Virginia Sports Medicine and Rehabilitation Institute), *Sweetbriar College*. Cat. I credits. Robert P. Nirschl, MD, 3801 N. Fairfax Dr., Arlington VA 22203, (703) 525-5542.

August 5-7
The EKG (Eastern Virginia Medical School), *Virginia Beach*. Office of CME, EVMS, Box 1980, Norfolk VA 23501.

August 10-13
Summer Retreat: Practical Issues in Primary Care (Medical College of Virginia), *Virginia Beach*. 17½ Cat. I

credits. Fee: $235. Office of CME, Box 48, MCV Station, Richmond VA 23298. (804) 786-0494.

August 11-13
Practice Management for the Emergency Physician (Virginia Chapter, American College of Emergency Physicians), Cavalier Hotel, *Virginia Beach*. 22 hrs Cat 1. Fee: $125-$400. Gwen E. Messler, PO Box 317, Highland Springs VA 23075, (804) 737-9433.

August 18-20
Practical Dermatology for the Primary Care Physician (Eastern Virginia Medical School), *Virginia Beach*. 14 credit hrs. Fee: $175. Office of CME, EVMS, PO Box 1980, Norfolk VA 23501.

August 18-21
Common Concerns of Plastic and General Surgeons (Eastern Virginia Medical School), *Virginia Beach*. Office of CME, EVMS, PO Box 1980, Norfolk VA 23501.

August 25-27
Reconstruction of the Breast (Medical College of Virginia), *Hot Springs*. 14½ credit hrs. Fee: $350. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

SERIAL LEARNING PROGRAMS

Colby College in Waterville, Maine, has presented continuing medical education courses since 1945, building a national reputation for courses of quality in a pleasurable summer milieu. Physicians and their families are accommodated on campus. The courses offered in 1983 are listed below; all are accredited for Category 1. For further information, write to Robert H. Kany, Colby College, Waterville ME 04901, or call (207) 873-1131.

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July 12-15	Surgical Techniques
July 14-15, 18-19	Computer Course
July 19-23	Infectious Disease
July 25-29	Emergency Medicine
July 25-29	Otolaryngology
August 1-4	Anesthesiology
August 1-5	Pulmonary Disease
August 7-11	Ophthalmology
August 8-12	Obstetricis/Gynecology
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August 14-19	Nuclear Medicine
September 12-16	Marine Medicine

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Industrial Medicine
E. Wilson Watts, Jr., M.D.

Family Practice
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Keith C. Edmunds, M.D.
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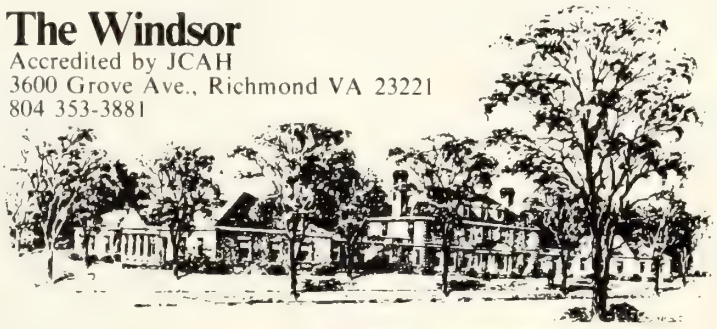

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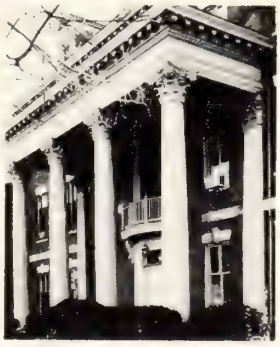

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Peter G. King, MD, Pathology, 3214 Lothian Rd., Fairfax VA 22031

Robert S. Korn, MD, Pediatrics, 4300 S. 35th St., Arlington VA 22206

David A. Schreiner, MD, Internal Medicine, 2001 Columbia Pike, Arlington VA 22204

Carol G. Mussenden, MD, Obstetrics/Gynecology, 12418 Hanger Rd., Fairfax VA 22033

Chesapeake Medical Society

Philip G. Leavy, Jr., MD, Emergency Medicine, 1125 Westover Ave., Norfolk VA 23507

Amante G. Legaspi, MD, Anesthesiology, 4424 Moosewood Dr., Virginia Beach VA 23462

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Anil Verma, MD, Internal/Pulmonary Medicine, 8346 Traford Lane, Springfield VA 22152

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Richmond Academy of Medicine

Paul S. Monroe, MD, Gastroenterology, 5855 Bremo Rd., Richmond VA 23226

Philip G. Schlobohm, MD, Psychiatry, PO Box 9127, Richmond VA 23227

Southwestern Virginia Medical Society

Stuart F. Mackler, MD, Orthopedic Surgery, PO Box 3768, Radford VA 24143

Tri-County Medical Society

Daniel K. Peak, Jr., MD, Internal Medicine, PO Box 755, Franklin VA 23851

Nellie M. Sankaran, MD, Pediatrics, PO Box 1815, Suffolk VA 23434

Virginia Beach Medical Society

Sheldon P. Fineman, MD, Anesthesiology, 1100 First Colonial Rd., Virginia Beach VA 23454

Wise County Medical Society

H.R. Ramamurthy, MD, Obstetrics/Gynecology, Wise Clinic, PO Box 17, Wise VA 24293

Nirmala Sreenivasan, MD, Pediatrics, Wise Clinic, Box A, Wise VA 24293

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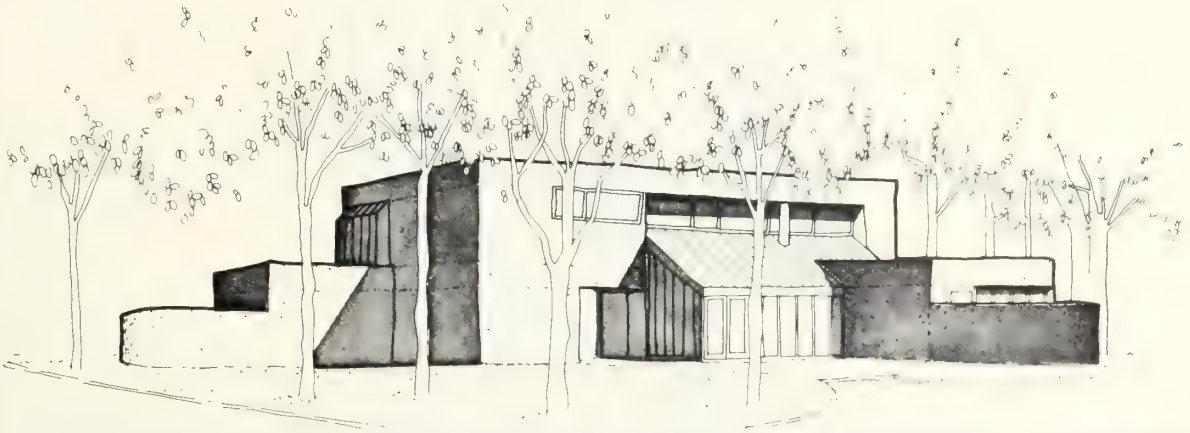

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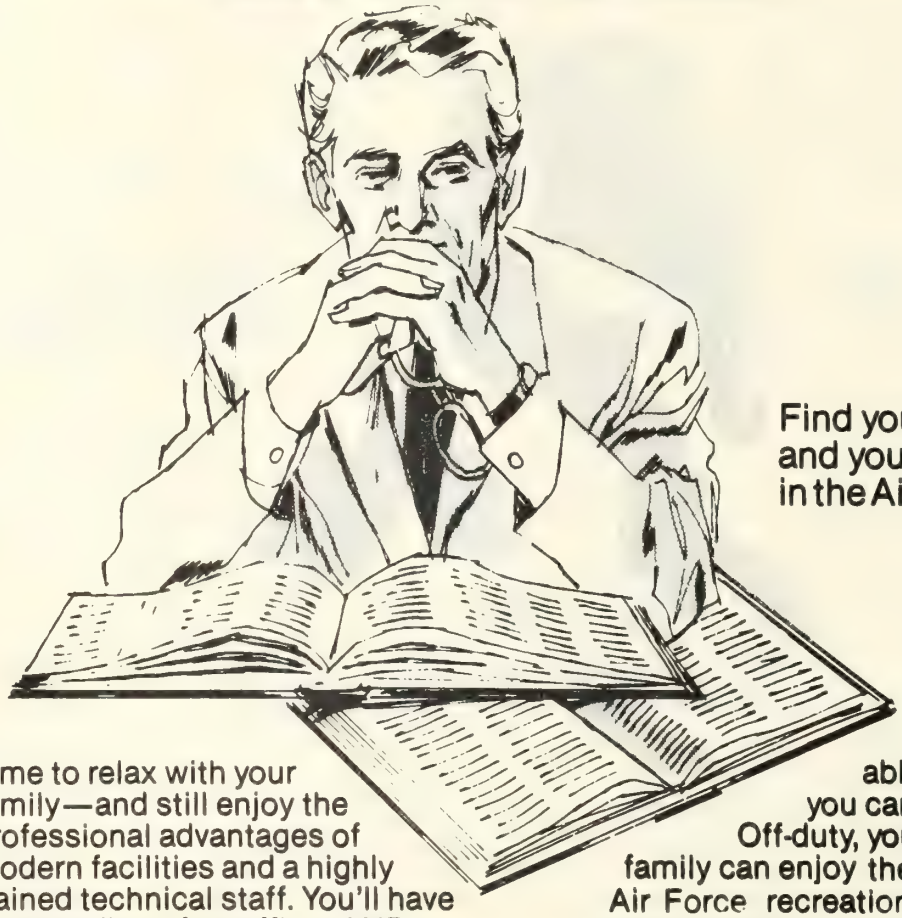

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Discontinuation of Valium (or Valrelease) is typically as smooth as its start in short-term therapy. However, Valium and Valrelease should be discontinued gradually after more extended treatment. As you diminish dosage, the built-in tapering action of Valium and Valrelease will help avoid rapidly recurring anxiety symptoms and symptoms of withdrawal, and will help ease the patient's transition to independent coping when therapeutic goals have been achieved.

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For a summary of product information, please turn the page.

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, impending or acute delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: relief of skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome. *Oral forms* may be used adjunctively in convulsive disorders, but not as sole therapy. *Injectable form* may also be used adjunctively in: status epilepticus; severe recurrent seizures; tetanus; anxiety, tension or acute stress reactions prior to endoscopic/surgical procedures; cardioversion

The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures

INJECTABLE *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins,* i.e., *dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated)

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear

INJECTABLE Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance

INJECTABLE Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia

In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported

Dosage: Individualize for maximum beneficial effect

ORAL *Adults:* Anxiety disorders, relief of symptoms of anxiety—Valium (diazepam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.)

For dosages in infants and children see below; have resuscitative facilities available

I.M. use: by deep injection into the muscle

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., *dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion*

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children *administer I.V. slowly*; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed Respiratory assistance should be available

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful

In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value

How Supplied:

ORAL Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10

Valrelease (diazepam/Roche) slow-release capsules—15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

INJECTABLE Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative

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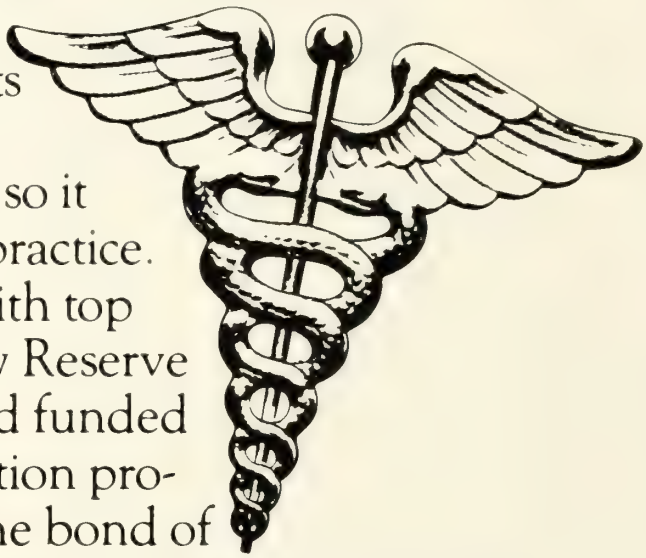

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The Adult Chemical Dependency Treatment Program deals with individual patient needs in a non-psychiatric approach that involves intense hospital care and patient/family education and counseling. Emphasis is on communication and non-chemical coping skills without the use of psychotropic drugs.

The Adolescent Chemical Dependency Treatment Program places special emphasis on the adolescent and the family, with therapy designed to help restructure the family unit and aid in the recovery of all. The full services of Peninsula Academy allow for continued education while the patient undergoes treatment.

The family program for Adolescent and Adult Chemical Dependency focuses on helping concerned individuals adjust to living with another's alcohol or drug problem. The program's non-psychiatric approach emphasizes family education to the problem, enhanced by individual and group therapy with an aim toward positive development of the whole self.

As with all Peninsula Psychiatric Hospital's programs, there are no eligibility restrictions with regard to race, religion, sex, residency or disability. For details on any of our Chemical Dependency Treatment Programs, telephone **Hampton (804) 722-2504, Norfolk (804) 622-5577, Richmond (804) 796-2016.**

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VIRGINIA
MEDICAL
JUNE 1983

Medicine Bitter and Sweet at the 1983 General Assembly

The Medical Society of Virginia won some and lost some at this year's legislative session, where measures affecting physicians dealt with such topics as "natural death," PPOs, malpractice awards, optometric therapy and "swing" beds.

Bob Brown

Newspaper and TV reporters beseige Dr. Read F. McGehee, Jr. (left) and Del. Bernard S. Cohen (D-Alexandria) as they leave a committee hearing on the Natural Death Act. Cohen sponsored the bill.

THIS YEAR'S SESSION of the Virginia General Assembly put more syrup than bitters into its medically-related legislative formula.

Not all of the mixture will go down easily for all of the state's physicians. Some is, at best, bittersweet. But the lobbyists who carry to Capitol Hill the messages from The Medical Society of Virginia concur that, all things considered, they got most of their messages through.

The medically-oriented legislative action that generated the most emotion and the most headlines during the January/February lawmaking orgy was passage of what was called the "Natural Death Act." That is a measure providing that a competent adult may make an oral or written declaration instructing the physician to withhold or withdraw life-prolonging procedures, or that person may designate another to make the treatment decision if he or she is diagnosed as suffering from a terminal condition.

For the steps Virginia physicians must take to invoke the protection the law affords them in these cases, see the next page.

The tone was set for passage of the act when the lawmakers at the Capitol received the report from a 13-member study group. The report strongly endorsed the concept of legislation on the subject, similar to that already passed in 13 other states and the District of Columbia.

Two Medical Society of Virginia members served on that study committee: Dr. Susan J. Mellette, medical oncologist at the Medical College of Virginia, and Dr. John W. Hoyt, medical director of the surgical intensive care unit at the University of Virginia Medical Center.

Two other MSV members were among those providing information for the study committee: Dr. William H. Regelson, like Dr. Mellette a medical oncologist at the Medical College of Virginia, and Dr. Read F. McGehee, pulmonary specialist in private practice in Richmond.

In the editorial section of this issue Dr. Mellette and Dr. Regelson comment on the Natural Death Act.

Joining the two Virginia physicians on the study committee were eight state legislators (five delegates, three senators); Nicholas A. Spinella and R. R. Young, Jr., representing the State Bar; and the Rt. Rev. David H. Lewis, Jr., suffragan bishop of the Episcopal Disocese of Virginia, representing the clergy.

Almost all those involved in the study committee and its public hearings appeared later before House or Senate committees when the General Assembly began deliberating the measure. In his appearance at a Senate hearing, Dr. McGehee emphasized that in supporting the bill he came as an advocate of patients, not doctors. He recalled that when he was graduated from medical school 22 years ago, there were no such things as respirators to keep people alive.

"But now," he added, "it is common to see patients kept alive for weeks and months"—to the diminishment of the patient's dignity and the detriment of the survivors' finances. He said he thought the bill's passage would make doctors less fearful of legal action and more attuned to patient, or family, desires for "death with dignity."

At that same hearing Allen C. Goolsby III, counsel for the Medical Society of Virginia, described the proposed legislation as the end result of the study committee's desire to "create a small safe harbor for people who prefer not to go to extraordinary measures to keep themselves alive."

Earlier, Dr. Regelson had addressed the study committee. With new technology and improved care, he said, people are being kept on machines when "the quality of life is very limited."

A "technocratic approach to the maintenance of life," had developed, he continued, with persons disabled beyond hope of recovery still emitting brain waves. Cessation of brain waves is a criterion used in the legal definition of death. He said that because of law suit threats, doctors are "in a very, very nervous state."

The bill, as passed, would seem to allay some of those fears. It reads, in part: "A health care facility, physician or other person acting under the direction of a physician shall not be subject to criminal prosecution or civil liability or be deemed to have engaged in unprofessional conduct as a result of withholding or the withdrawal of life-prolonging procedures from a patient with a terminal condition in accordance with this Act."

Two Medical Society of Virginia members came to the Capital Building to make known their express displeasure with the Natural Death Act. They were Dr. Robert J. Fierro, Richmond gynecologist, and Dr. Henry B Larzelere, Lynchburg surgeon.

"If a lawyer wants to sue you, you will be sued," warned Dr. Fierro.

Dr. Larzelere doubted that "50 doctors in the State of Virginia have even read the bill." He added that he wasn't at all certain that The Medical Society of Virginia's formal decision not to oppose the act represented the majority thinking of the state's physicians.

It remained, however, for still another physician,

CLIP AND KEEP FOR REFERENCE

Physician's Guide to the Natural Death Act

WHEN it goes into effect this coming July 1, the Natural Death Act will offer a statutory "safe harbor" for the Virginia physician who complies in good faith with its provisions. These are the steps the law requires and the definitions it makes:

1. A patient makes a declaration directing the withholding or withdrawing of life-prolonging procedures in the event of a terminal condition. If the declaration is written, it must be signed by the patient and two witnesses, neither of which may be the patient's spouse or blood relative but one of which may be the physician. If the declaration is oral, it is valid only 1) if made after the patient has been diagnosed as having a terminal condition and 2) if it is made in the presence of a physician and two witnesses, neither of which may be the patient's spouse or blood relative.

2. If the patient has made a valid declaration and is competent, the attending physician must then certify in writing that the patient has a terminal condition before withholding or withdrawing a life-prolonging procedure.

The law defines "terminal condition" as "a condition caused by injury, disease or illness from which, to a reasonable degree of medical certainty, (i) there can be no recovery and (ii) death is imminent."

"Life-prolonging procedure" is defined by the Act as "any medical procedure, treatment or intervention which (i) utilizes mechanical or other artificial means to sustain, restore or supplant a spontaneous vital function or is otherwise of such a nature as to afford a patient no reasonable expectation of recovery from a terminal condition and (ii) when applied to a patient in a terminal condition, would serve only to prolong the dying process. 'Life-prolonging procedure' shall not include the administration of medication or the performance of any medical procedure deemed necessary to provide comfort care or to alleviate pain."

4. If the patient has made a valid declaration but is "comatose, incompetent, or otherwise physically or mentally incapable of communication," the attending physician must certify in writing that the patient has a terminal condition and must also have one other physician examine the patient and do likewise before withholding or withdrawing a life-prolonging procedure.

5. In the case of a patient who is comatose, incompetent or otherwise physically or mentally incapable of communication and who has not made a written or oral declaration, the Act designates a person or persons with whom the attending physician must consult and agree prior to withholding or withdrawing a life-prolonging procedure.

Listed in order of priority, these designated persons are 1) any guardian or committee judicially appointed on behalf of the patient; 2) anyone designated in writing by the patient to make the treatment decision; 3) the patient's spouse; 4) an adult child of the patient or a majority of the children who are reasonably available for consultation; 5) the parents of the patient; 6) the nearest living relative of the patient.

If no one in the first category is "reasonably available, willing and competent to act," the physician may go to the next priority, and then the next, until an appropriate person is located.

But when the physician must turn to any of the family members in the last four categories, the physician 1) must insure that there are two witnesses present when the treatment decision is made with those family members, and the patient's spouse and blood relatives may not serve as either of these witnesses, and 2) must get consent from two persons in any of the last four categories if two are reasonably available.

Two points should be emphasized. First, the Act does not apply to patients who are under 18 years of age. Second, if a physician does not want to direct the withholding or withdrawing of life-prolonging procedures as authorized by this Act, he has no obligation to do so; he must, however, offer to transfer the patient to another physician.

The Act, which does not eliminate any immunity a physician already may have under the common law, is to be codified as Virginia Code §§ 54-325.8:1–54-325.8:13.

Attorneys Sandra Kramer and Allen C. Goolsby III developed this legal information for VIRGINIA MEDICAL.

Dr. John C. Buchanan, state senator from Wise County and the only physician in Virginia's 140-member legislature, to articulate the most vivid testimony against the bill: "The only good I say about it," he said to a Senate committee, "is that the people who support it have good intentions."

He said he had seen "many people die during my years of practice, but I have seen patients who did recover and were able to go home . . . I've been fooled and I'm sure other doctors have been; I'm

For more medicine and the law, see page 348.

afraid that is just getting a foot in the door and in the next year or so we'll be getting a bill providing for euthanasia."

Dr. Buchanan repeated on the Senate floor much of what he had said in committee. But at neither site did his sentiments prevail. The Senate Committee on Education and Health, shortly after he completed his anti-bill arguments, approved the bill by 10-5 vote. In the Senate itself, the vote was 21-18 in favor. The legislator-physician's speech there may have persuaded some members, but it did not persuade enough to defeat the bill.

One section of the act addresses itself to "mercy killing", or euthanasia, stating that nothing in the act "shall be construed to condone, authorize or approve mercy killing or euthanasia, or to permit any affirmative or deliberate act or omission to end life other than to permit the natural process of dying."

The act provides felony penalties for anyone who forges or destroys a statement of authority to cut off life-support systems. It says also that withdrawal of life-support systems "shall not for any purpose constitute a suicide."

WHILE the Natural Death Act drew the headlines and the impassioned oratory, there was another piece of legislation that went virtually unheralded in the daily media as it was taking shape. That was the act enabling establishment of preferred provider organizations in Virginia.

"That, I firmly believe, is the most significant—or will turn out to be the most significant legislation affecting the health field at this year's session," said Goolsby, who is one of two men who bear most of The Medical Society of Virginia's lobbying responsibilities. The other is Willard C. Osburn, director of legislative activities for the Society.

VIRGINIA MEDICAL's May issue carried a cover story on the first PPO to be enabled by the legislation.[1] It is called "Keycare," it is slated for the Richmond-Petersburg area, it is offered by Blue Cross Blue Shield of Virginia, and it follows the PPO formula: "Preferred provider" hospitals and physicians will agree to tender services at contracted rates; subscribers will help pay the bills through deductible clauses and copayments.

The plan is scheduled to begin operation on or about July 1 with at least five hospitals—and an undetermined number of doctors—participating. Ultimately, there may be other Keycares over Virginia. The Tidewater area has been tentatively selected as the site for the next one, to open early next year.

THE bitterest syrup the Assembly mixed up for the medical profession was its approval of a bill permitting optometrists to administer certain diagnostic drugs to their patients. That bill was opposed by the Society's lobbyists, as similar proposed legislation had been during the past five years. Heretofore the measure had either been beaten or vetoed by the governor. This year, though, there was neither defeat nor veto.

The state's ophthalmologists vigorously opposed the legislation, arguing that the non-medical optometrists should not be permitted to use various dilating and cycloplegic drugs, termed diagnostic drugs. They were especially opposed to the optometrists' use of pilocarpine, which they termed a therapeutic drug for the treatment of glaucoma.

Said Will Osburn of its passage, "Well, except for that, we had a pretty good year."

MALPRACTICE, HSAs and "swing" beds were among the concerns of other offered measures at the 1983 General Assembly. Some passed, some didn't.

The one on malpractice increased the cap on malpractice suit awards from $750,000 to $1 million, and it passed. But, Goolsby rationalized, the change wasn't particularly bitter medicine.

"We'll just have to figure that one as a cost-of-living increase," he said. "Even with the increase, the cap does give some peace of mind to insured doctors that no one's going to come and take their home and car away from them." He noted that malpractice policies have coverage limits of $1 million.

Backed by the Society and passed by the Virginia lawmakers was a bill granting civil court immunity to physicians and dentists called upon to investigate drug or alcohol addiction among their professional

colleagues. This measure relates to the growing effort of both professions to police their ranks. The Medical Society of Virginia recently established the Physicians Health and Effectiveness Commitee "to assist in the discovery and rehabilitation of physicians whose professional effectiveness is jeopardized by special and continuing problems."

Some sweetening came in bills that were killed by the Assembly, most of them at the committee level of the legislative process, before they even reached floor-vote stage. Among them, some of which were opposed actively and some passively by Osburn and Goolsby:

- An attempt to add a nursing home administrator and a podiatrist to the nine-member State Board of Health.
- An effort to help fund five health planning agencies by increasing license fees for doctors, nurses, and allied health professionals, such as osteopaths, psychologists and pharmacists. That same bill also would have imposed a hospital-bed user's fee (like city fathers sometimes impose on motel rooms in resort areas).
- Proposals for the licensing of occupational and respiratory therapists (another addition to the bureaucracy).

The Medical Society of Virginia, through its spokesmen, favored one bill that passed despite vigorous opposition from nursing home entrepreneurs. That legislation permits hospitals, subject to certain restrictions, to use "swing," or unused, beds as nursing home beds. As an example of the restrictions, the use would be for a maximum of 30 days for any patient.

Also supported by the Society was a bill that made modest language changes designed to increase the likelihood that anyone who knowingly prescribes or dispenses prescription drugs for nontherapeutic purposes will be prosecuted criminally. The bill, which was strongly supported by the Attorney General, also will make it easier to transmit incriminating information from regulatory boards to law enforcement agencies. —C. L. SHIRES

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Dr. Holland declares for the Virginia Senate

Though it's a long long time from now till the November election, there is a real possibility that a member of The Medical Society of Virginia, Dr. Clarence A. Holland, will become a member of the Virginia State Senate.

The Virginia Beach general practitioner is the Democratic candidate to fill a Senate seat created during the re-districting of the General Assembly that took place in 1981. If elected, he would be the only Medical Society of Virginia member seated in the legislature. Another physician-senator, Dr. John W. Buchanan of Wise County, is not an MSV member.

Dr. Holland, who turns 54 late this month, is a native of Isle of Wight County and was graduated from Hampden-Sydney College and the Medical College of Virginia. He served on the Virginia Beach City Council 12 years, two of them as Mayor.

Optimism about Dr. Holland's election was expressed by another Virginia Beach physician, Dr. Frederick K. McCune, The Medical Society of Virginia's second vice president and chairman of its Public Relations Committee.

Without disclosing his own political party persuasions, Dr. McCune said this: "I think most Virginia Beach physicians will support him strongly and won't worry about his party affiliations. We desperately need someone up there at the Capitol with political clout and Clancy has got political clout. I think without a doubt he will take it.

"Besides, he's got politics in his blood, he's a proven good campaigner, and it certainly is anticipated that he will win the endorsement of the Virginia Beach Medical Society."

Dr. McCune's reference to "politics in the blood" hearkens to two things: Dr. Holland's father, Shirley T. Holland, served for 20 years in the Virginia House of Delegates and was a stalwart member of the conservative Democratic organization, and his brother, Richard J. Holland, an Isle of Wight County banker, is seeking reelection to the Virginia Senate. (There is another Holland in the State Senate, Edward M. (D-Arlington), but he's no relation.)

Dr. Holland, an insider in Virginia Beach Democratic politics, headed the gubernatorial campaign in the resort city for Charles S. Robb. He said he was as yet not certain whether Robb would assume any active role in his own campaign. —C.L.S.

For Divers, a Life-Saving Network

SCUBA DIVING is a popular sport in Virginia. In the Richmond area alone there are 3,000 certified sport divers, and 500 more are certified each year. Courses in scuba diving are offered throughout the state at colleges, YMCAs, dive shops, recreation centers and swim clubs. The graduates of these courses practice their new skills in Virginia's quarries, rivers and lakes and in Chesapeake Bay and the Atlantic Ocean.

Their dives are often in excess of 100 feet, where the water is cold and the visibility poor, and therein lies the potential for serious accidents, for example, arterial gas embolism and decompression sickness. Management of these two conditions requires the use with all possible speed of a recompression chamber. Management en route to the chamber includes basic life support, oxygen, the Trendelenburg position, fluids, aspirin, and steroids.

In any situation suggesting an underwater diving accident, the primary question is "Did the diver breathe compressed air under water?" If the answer is "yes," or if the diver is unconscious, he or she must be regarded as a diving accident victim.

This advice and much other clinical information on the causes, signs, symptoms and management of underwater accidents appears in the Underwater Diving Accident Manual, which was prepared under the auspices of the National Diving Accident Network and edited by G. Yancey Mebane, MD. For a copy, write to the Diving Accident Network at Duke University Medical Center, Durham, North Carolina 27710. The cost is $3.

The manual is but one of the services of the National Diving Accident Network, which was formed in 1981 to assist in the treatment of diving accidents. The Network provides a 24-hour emergency call number, 919-684-8111. A physician is on call at that number to supervise referral to the nearest decompression chamber and to give advice on the emergency management of the diving accident victim.

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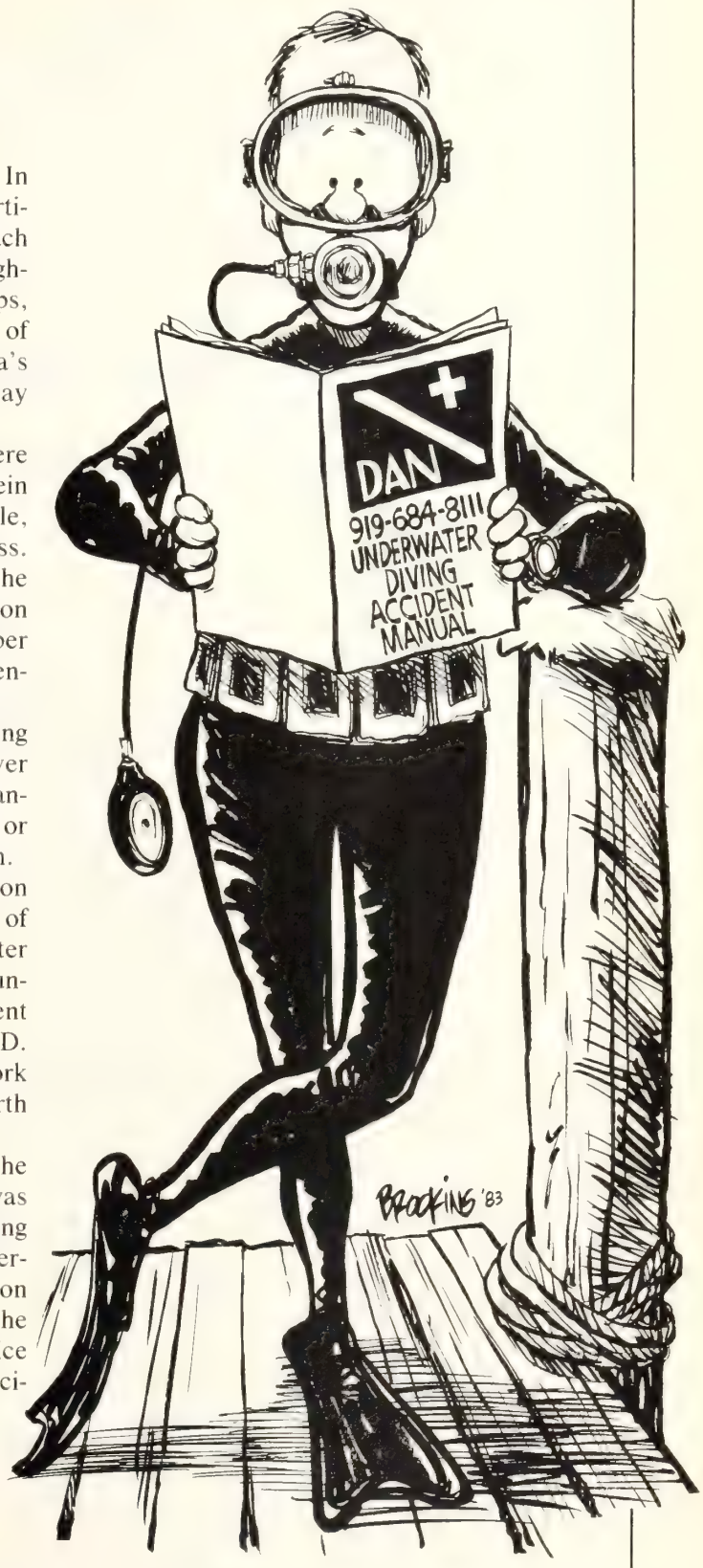

Measles and Rubella in Virginia: Age Shifts Bring New Problems

Gregory F. Hayden, MD, *Charlottesville, Virginia*

Due to widespread immunization of infants and children, the greater incidence of measles and rubella now occurs among adolescents and young adults. The author suggests ways in which the physician can help solve this problem, for instance, more aggressive vaccination of post-pubertal females.

The widespread use of live, attenuated viral vaccines for measles and rubella has had a dramatic impact upon the epidemiology of these two diseases. Vaccination has been immensely successful in reducing the morbidity and mortality associated with these diseases within Virginia and nationally, but several new problems have arisen.

One particularly troublesome change is the increasing percentage of cases of measles and rubella among adolescents and young adults.[1,2] Over the past five years, reported measles and rubella have decreased markedly among young children due largely to the enactment and vigorous enforcement of school immunization laws,[3] but similar decreases have not been noted among adolescents and young adults, so that this slightly older segment of the population now accounts for a much larger percentage of reported cases. A recent measles outbreak in Lee County, Virginia, for example, was traced to a 15-year-old foreign-exchange student and involved primarily adolescents and young adults.[4] This upward change in the age distribution of measles and rubella cases has important implications for the continuing control of these diseases in the future.

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Measles

Reported measles is now at an all-time low. Only 14 cases were reported in Virginia during 1982, down more than 99% from levels during the prevaccine era (Table 1), and national trends are similar.[5] Concomitant with this decline in reported cases, the age of persons with reported measles has risen sharply. In 1972-1973, over half the measles cases reported in Virginia occurred in children under 5 years of age, and 80% of cases were in children under age 10 years (Table 2). But in 1980-1982 fully 73% of measles cases reported in Virginia occurred in persons *over* age 10 years, a nearly complete age reversal from the earlier era.

This upward shift in the age distribution of measles cases has prompted speculation as to whether vaccine-induced immunity has waned and left adolescents unprotected years after vaccination. Many adolescents developing measles in recent years had

previously received measles vaccine but often not in optimally effective ways, e.g., vaccination before one year of age.[6] Some measles cases have been reported among adolescents previously vaccinated in an acceptable fashion, but such occurrences have generally not exceeded the number expected on the basis of the anticipated 2-5% rate of failure to seroconvert. Serologic studies have generally shown excellent persistence of measles antibodies for at least ten years after vaccination, and vaccinees who reverted to seronegative after successful vaccinations have demonstrated booster-type immune responses when revaccinated, suggesting functional immunity.[7]

In many other recent instances, adolescents with measles had never received measles vaccine, so it appears that the primary reason for the recent prominence of adolescent measles has not been vaccine failure but either failure to vaccinate or, in some instances, failure to vaccinate correctly.

The upward shift in the age of reported measles cases poses a practical problem for the practitioner. The Virginia school immunization law is to change in July of this year to include not only school enterers, usually kindergarten and first grade, but all school attenders in grades kindergarten through 12.[5] This law makes it necessary to determine which middle- and high-school students are adequately protected against measles and which ones should be vaccinated or revaccinated.

The history of measles vaccination in this country is not straightforward. Two different measles vaccines were licensed in 1963. One was the live, attenuated Edmonston B strain, which was very immunogenic but not adequately attenuated, so that its recipients often developed fever and rash. For this reason, this vaccine was often given with an accompanying dose of gamma globulin, and in accordance with early recommendations, was often given as early as 9 months of age. The other initial product was a killed virus vaccine often given in multiple, sequential doses. The syndrome of atypical measles was soon recognized among recipients of this vaccine, which had faded from use by 1968. A further attenuated, live vaccine became available in 1965, and remains in use today; unfortunately, some physicians continued to administer an accompanying dose of ISG with this weaker, further attenuated vaccine. The current vaccine is not sufficiently immunogenic to be given with simultaneous ISG nor to be given under one year of age, when low levels of transplacentally-acquired measles antibody can interfere with viral replication and vaccine "take".

Table 1. Reported Cases of Measles and Rubella in Virginia.

	Measles	Rubella
Pre-vaccine era*	9,886	759
1977	2,774	585
1978	2,837	247
1979	288	208
1980	339	42
1981	18	9
1982	14	12

* Yearly average for three-year interval preceding introduction of respective vaccines, i.e., for measles, 1960-1962, and for rubella, 1966-1968.

Table 2. Proportion of Reported Cases of Measles and Rubella in Virginia According to Age Group.

	Percentage of Measles Cases		Percentage of Rubella Cases	
Age (years)	1972-3 (N = 501)*	1980-2 (N = 371)	1972-3 (N = 613)**	1980-2 (N = 63)
<5	55	12	28	22
5-9	25	14	19	6
10-14	8	28	20	19
15-19	6	32	22	27
20+	6	13	12	25

* Percentage based upon 127 cases in persons of known age
** Percentage based upon 112 cases in persons of known age

By the late 1970s, it appeared that interference by maternal antibody was occurring in a very small percentage of vaccinees even at 12 months of age, so it was recommended that routine measles vaccination be postponed until age 15 months. The difference in rates of seroconversion at 12 versus 15 months appears so small, however, that it is unnecessary to revaccinate children who have previously received the live, further attenuated vaccine between 12 and 14 months of age.[8]

In reviewing the immunization records of adolescents and young adults, one must therefore seek out not only those who have never received measles vaccine, but also those who were vaccinated in a less than optimally effective manner. Some practical suggestions for making the determination of probable measles immunity or susceptibility are outlined in the accompanying box. Persons who received the killed measles vaccine may develop local or systemic reactions when revaccinated with the live, further attenuated vaccine, but the risk of developing atypical measles if exposed to wild virus is felt to justify the risk of less serious vaccine reactions. Selective serologic testing for measles antibodies may identify some adolescents whose initial live measles vaccination was improper or uncertain but who now have serologic evidence of measles immunity. Such children are probably immune and do not require revaccination. There is,

INDEX TO MEASLES IMMUNITY

Consider as **immune** children with documented a) physician-diagnosed measles, or b) live measles vaccine on or after the first birthday. Persons born 1957 or earlier are generally considered to be immune regardless of vaccination history.

Consider as **susceptible** children who received a) only killed measles vaccine; b) a rapid (<3 mos) killed-live sequence; c) live vaccine before one year of age; d) live, further attenuated vaccine with simultaneous ISG.

Consider as **potentially susceptible** children whose immunization status cannot be determined adequately, e.g., those who received an unspecified measles vaccine before 1968.

Serologic testing may be helping in determining immunity in selected cases.

however, no known risk of revaccinating persons who are already immune, so serologic testing before revaccination is not mandatory.

Rubella

The last major rubella epidemic in this country occurred in 1964, and reported rubella is at all-time low levels.[9]

Only 12 cases were reported in Virginia during 1982, an almost 99% reduction from levels during the pre-vaccine era (Table 1). Since many cases of rubella are not clinically apparent, not diagnosed, or not reported to the Health Department, reported levels may severely underestimate the actual numbers of cases occurring, but the recent decrease in reported rubella activity is nonetheless striking and national trends are again similar.

But, as levels of reported rubella have decreased, the average age of persons with rubella has increased (Table 2). In 1972-1973, 47% of reported rubella cases in Virginia occurred in children under 10 years of age and only 34% of cases occurred in persons over age 15 years. In contrast, only 28% of reported cases during 1980-1982 occurred in children under 10 years of age and 52% occurred in persons over age 15 years. This upward shift in the age distribution of reported cases has several important implications for the continuing control of this disease.

First, it appears that more aggressive vaccination of post-pubertal females will be necessary for further reductions in rubella activity in the years ahead. The wisdom and feasibility of this approach depend in part upon whether the attenuated rubella vaccine virus may itself be teratogenic. The vaccine virus can cross the placenta and infect the fetus of a susceptible vaccinee, but whether it can damage a fetus remains uncertain.[10] Over 140 infants have now been born to susceptible vaccinees. A few of these infants have had serologic evidence of congenital rubella infection, but all have been normal clinically. The observed risk of malformation has been zero, and the actual risk is estimated to be no greater than 3%, much less than the comparable risk of malformation following early gestational infection with wild rubella virus. On account of this favorable experience, many physicians have become willing to vaccinate post-pubertal females. It must be emphasized, however, that pregnancy remains a contraindication to rubella vaccination, and postpubertal women who are vaccinated should be advised to avoid pregnancy for 2-3 months after vaccination. The rubella vaccine virus is non-transmissable, so that the 15-month-old infant of a rubella-susceptible, pregnant woman may be vaccinated.

Another problem related to the increased prominence of rubella among adolescents and young adults involves the durability of vaccine-induced immunity. Will a female infant vaccinated at age 15 months remain protected through her child-bearing years or will she require a booster shot during adolescence? The later pertinent information is reassuring. In the largest study, over 98% of vaccinees who initially seroconverted have remained seropositive ten years later.[11] In addition, several vaccinees who had reverted to seronegative have been revaccinated, and all showed booster-type immune responses suggesting functional immunity. These data are particularly comforting since smaller, earlier studies had observed a more rapid waning of vaccine-induced immunity and also because these favorable results were obtained with three of the earlier rubella vaccines. The vaccine in current use (RA 27/3) is more immunogenic than these previous strains and more likely to induce durable immunity without a significant increase in side effects.[12] At least for the present, it appears that booster shots for adolescent females will not be necessary.

One final problem related to the upward age shift in reported rubella involves a recent development in nosocomial rubella. The traditional concern in this regard has been transmission of rubella from an infected patient to a susceptible pregnant employee. Female hospital employees of child-bearing age have therefore been encouraged or required to have a rubella titer performed and to be vaccinated as appropriate for their own protection. In the past few years, however, there have been several instances of transmission in the other direction, from an infected young adult hospital employee to a susceptible pregnant patient.[13-15] In one outbreak, for

example, a male obstetrics resident with rubella exposed several hundred pregnant patients in a prenatal clinic. Because of these episodes, some authorities now recommend that all employees, male and female, in contact with pregnant patients have a rubella titer performed and be vaccinated as appropriate for the safety of the patients.

Summary

Despite dramatic decreases in reported measles and rubella activity in Virginia over the past five to ten years, several problems persist. Routine immunization of infants and young children now protects these groups, and a greater percentage of reported cases of both measles and rubella now occurs among adolescents and young adults. This trend has several important implications for the practitioner. For measles, it lends urgency to the task of reviewing the immunization records of young adults to determine who may require measles vaccination or revaccination. For rubella, it places renewed emphasis on the vaccination of post-pubertal females, the durability of vaccine-induced immunity, and the possible need for new hospital policies concerning nosocomial rubella.

Ms. Joanne Butler, nurse epidemiologist for the Virginia Department of Health, provided information about the age distribution of reported measles and rubella cases. Mrs. Marjorie Hampton prepared the manuscript.

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Virginia's new school immune law starts July 1

Virginia's comprehensive new law on the immunization of school children takes effect this coming July 1 for the 1983–1984 school year.

The law requires that all pupils attending public, private and parochial schools and licensed day-care centers be vaccinated against contagious diseases. This includes pupils in grades kindergarten through senior in high school, up to and including age 20.

Pupils must provide documentation showing the day, month and year they received inoculations for diphtheria, pertussis, tetanus, poliomyelitis, rubeola, rubella and mumps. To support a medical reason for exemption from this requirement, a physician must provide written certification that administration of the vaccines has been withheld because it would be detrimental to a pupil's health. To apply for religious exemption, the parent or guardian must make affidavit attesting to conflict of immunization with a pupil's religious tenets. In either category, the State Department of Health may order the child's exclusion from school in the event of an outbreak of contagious disease.

Symmetrical Osseus Metastases from Renal Cell Carcinoma: Case Reports

Richard Whitehill, MD,
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Charlottesville, Virginia

It is well known that renal cell carcinoma behaves in a most bizarre fashion in respect to its growth.[1] Osseus metastases from renal cell carcinoma occur in 34% to 50% of cases with metastatic disease.[2] Usually these bony metastases involve either the axial skeleton or the proximal portion of the long bones, sometimes bilaterally,[3] but there is a report of simultaneous metastases of renal cell carcinoma to the finger and the toe in the same person.[4] There is a report of the appearance of a metastasis from renal cell carcinoma some five years following nephrectomy.[2] Finally, there are two reports of spontaneous regression of bony metastases following nephrectomy.

This report emphasizes another unusual aspect of renal cell carcinoma, that is, its ability to produce *symmetrical* skeletal metastases.

Case 1

H.T., a 64-year-old white male, presented to us with bilateral upper arm pain. Three years prior to admission the patient underwent an open biopsy of a lytic lesion of his left proximal humerus, which was read as undifferentiated carcinoma. This lesion was then treated with an unknown dose of radiotherapy. He did well for 2½ years but over the six months prior to presenting to us had progressively increasing bilateral upper arm pain.

X-ray revealed symmetrical lytic lesions involving both proximal humeri as shown in Figure 1. An intravenous pyelogram showed a left renal mass. A routine laboratory evaluation, including a CBC, sed rate, calcium, phosphorous, acid phosphatase and serum protein electrophoresis, were within normal limits.

Under fluoroscopic control the patient underwent percutaneous fixation of both impending humeral fractures with a combination of methylmethacrylate and rush rods. Marked hemorrhage was encountered while biopsing each lesion, but this was easily controlled with impaction of the methylmethacrylate (Fig. 2). The biopsy obtained three years previously as well as the current biopsies were read as renal cell carcinoma.

Case 2

C.C., a 23-year-old white female, first presented to us in September 1976 with a chief complaint of abdominal pain. Arteriography revealed a left renal mass, and the patient underwent a left nephrectomy, at which time a renal cell carcinoma was diagnosed.

The patient did well until January 1978, when she noted a palpable left cervical lymph node. Biopsy of the lymph node revealed metastatic renal cell carcinoma. Shortly thereafter the patient began complaining of bilateral shoulder pain. X-rays revealed bilateral symmetrical lytic lesions involving both scapulae as shown in Figure 3.

Although the patient has been treated with a combination of chemotherapy and radiotherapy, she has recently been hospitalized with multiple new bony metastic lesions.

Summary

These two case reports emphasize another of the somewhat bizarre behavioral aspects of renal cell carcinoma metastatic to the skeletal system, i.e., its ability to produce symmetrical metastatic lesions. We suggest that when one bone metastasis is identified, others in symmetrical locations should be sought.

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Submitted 7-1-82.

Figure 1, Case 1. X-rays of humeri of 64-year-old male who presented with bilateral arm pain.

Figure 2, Case 1. X-rays of bilateral humeral lesions shown in Figure 1 following biopsy and fixation.

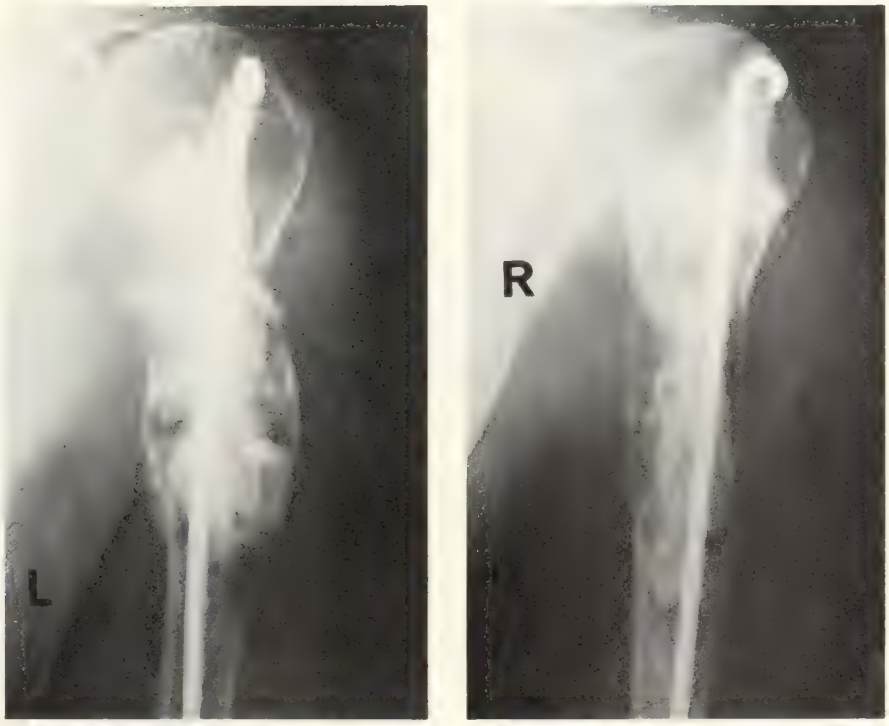
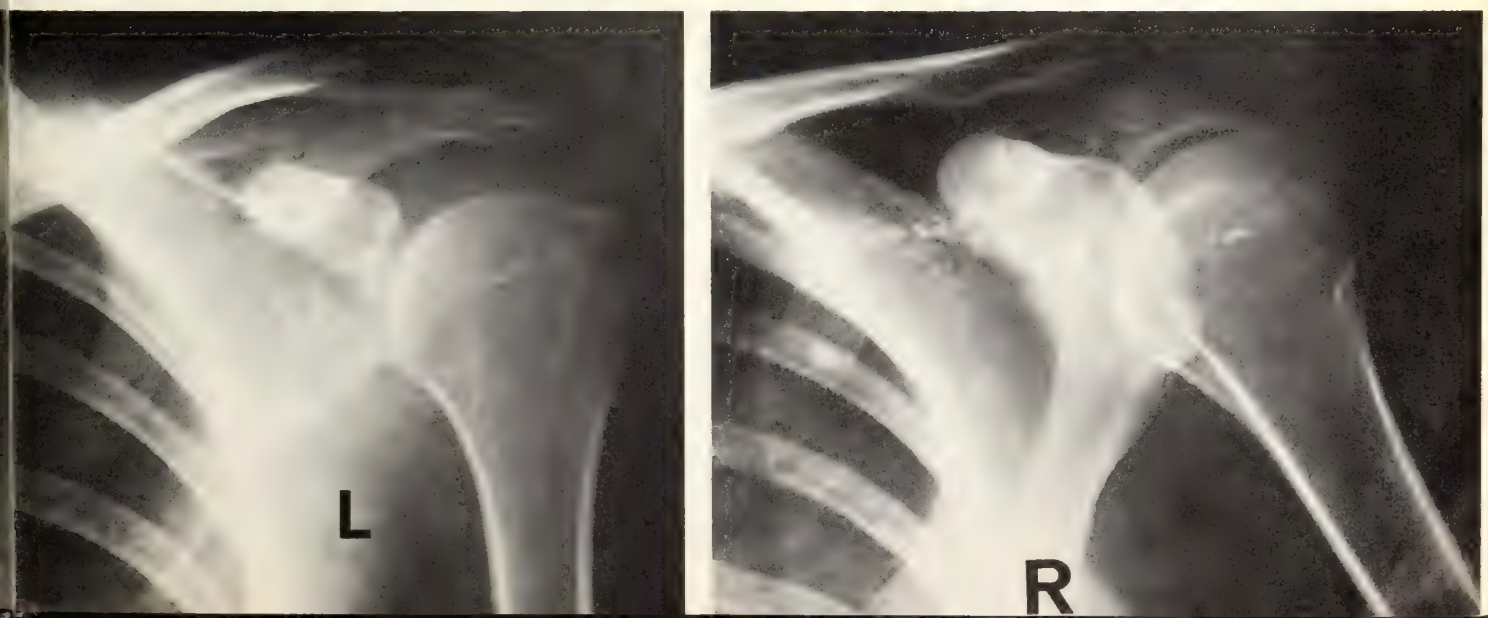


Figure 3, Case 2. Below, x-rays of 23-year-old female with renal cell carcinoma showing lytic lesions involving lateral aspects of both scapulae.



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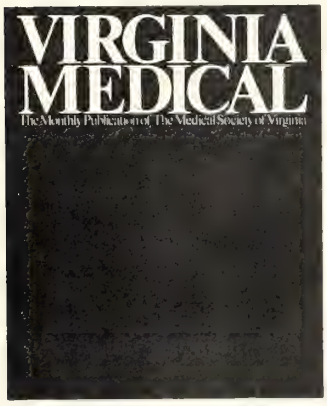

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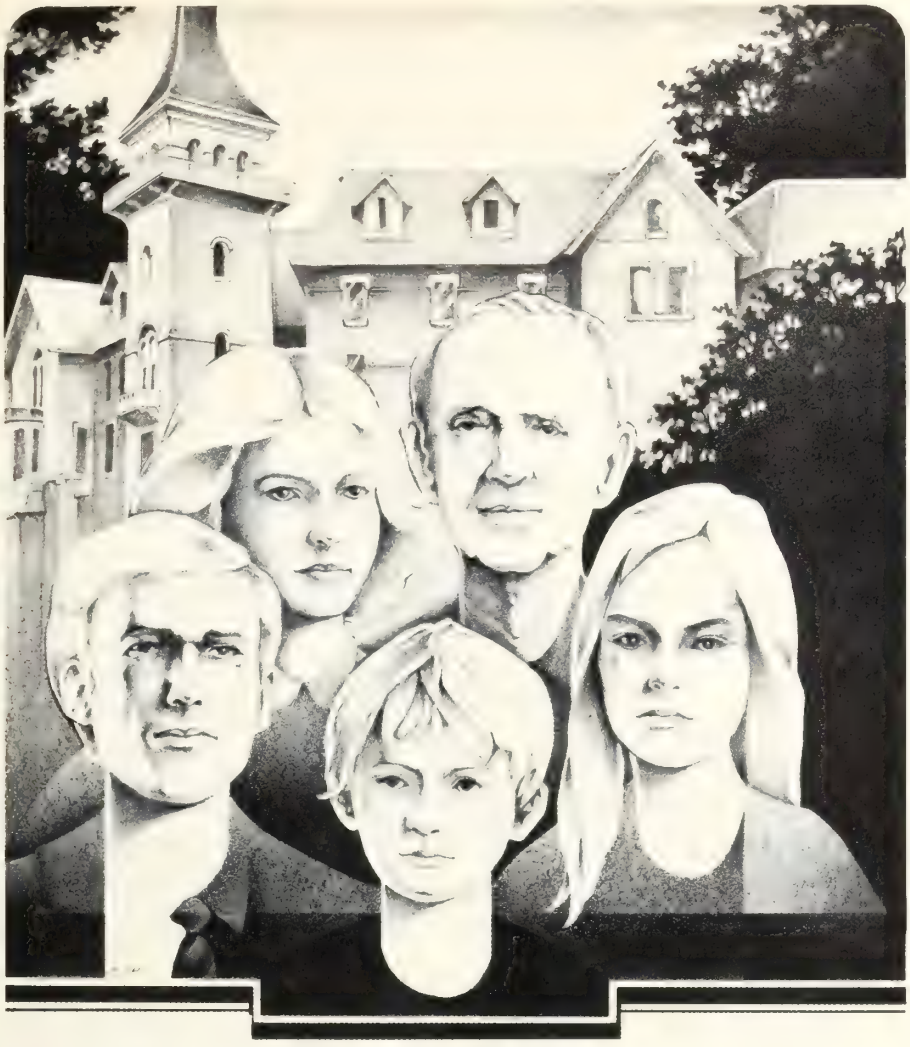

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Trait Marker for Mental Illness: Dexamethasone Suppression Test

Jonathan G. Solomon, MD, *Hampton, Virginia*

In the differential diagnosis of primary affective disease, particularly depression and dementia, the dexamethasone suppression test's measurement of cortisol response appears to make a helpful contribution. The author reviews the literature and offers three case reports illustrating the test's value.

THE employment of neuroendocrine tests in the search for trait markers for mental illnesses has emerged as an important feature of biological psychiatry.[1] One such neuroendocrine test is the dexamethasone suppression test (DST). Excessive activity of the hypothalamic-pituitary-adrenocortical (HPA) axis has been demonstrated in many patients with primary affective disorder.[2-9] As noted in a recent article,[2] evidence for overactivity in patients with severe depression includes reports of elevated plasma,[3,4] urinary,[6,8] and cerebrospinal fluid[10] levels of corticosteroids with disruption of the 24-hour cortisol secretory pattern,[4] with more cortisol secreted during the night and a larger number of secretory episodes.

In recent years the most frequently employed test to evaluate HPA activity in depressed patients has been the DST.[2,11-17] The patient is given an oral tablet of 1 mg of dexamethasone at 11 PM, and serum cortisol levels are obtained the following day at 4 PM and 11 PM. Most studies have reported escape from suppression of cortisol after the administration of dexamethasone in from 25-50% of patients with primary affective disorder.[11-17] The ability to suppress serum cortisol concentration in response to oral dexamethasone is impaired in many depressed patients, who are considered DST-positive or DST-nonsuppressors. The strategy of employing the overnight DST to assess cortisol responses seems promising.

The DST can be particularly helpful in the differential diagnosis of pseudodementia[18,19] and organic brain syndromes because elderly patients with depression may present with "organic" findings suggestive of dementia. In a previous communication,[20] I focused attention on the recognition and treatment of remediable causes of dementia and included the differential diagnosis of depression and pseudodementia (depression appearing as cognitive impairment rather than as a mood disorder). In this report the focus is on the use of the overnight DST

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in differential diagnosis, with special reference to the clinical dilemma posed by elderly depressed patients who may suffer from dementia, depression, or both disorders.

Recent reports highlight the efficacy of using the DST in differentiating pseudodementia from true dementia. Rudorfer and Clayton[21] described the use of the DST in assessing diagnosis, treatment response,[22-24] and prognosis in patients with primary depressive illness. They also claimed that response to somatic treatment of depression is generally good in patients who were DST-nonsuppressors.[22-25] They presented a case of a patient who seemed "organic," but thorough evaluation of the patient was negative, including CBC, urinalysis, SMA-6, SMA-14, thyroid studies, B_{12} and folate levels, urine toxic screen, EEG, CT scan of the head, and lumbar puncture. A DST revealed an early escape from suppression with appropriately low post-dexamethasone cortisol levels in the morning following dexamethasone administration, followed by rising serum levels of cortisol in the late afternoon and evening to 31.2 mcg% and 14.5 mcg% at 4 PM and 11 PM, respectively. The early escape from cortisol-suppression characterizes severe depression. Serial DST determinations during the course of electroconvulsive therapy administered to the patient correlated with the observed clinical improvement.

McAllister et al[26] reported on two patients with severe depression and pseudodementia. One patient was a 75-year-old woman with diabetes mellitus, a diagnosis of organic brain syndrome, and cerebral cortical atrophy on the CT scan of the brain. The other patient was a 72-year-old man with cognitive impairment and catatonia and a history of a catatonic episode 25 years earlier. His CT scan of the brain revealed a probable old infarction in the distribution of the right middle cerebral artery and raised the possibility of a new infarction in the distribution of the left middle cerebral artery. After repeated neuropsychiatric studies consistently failed to confirm a specific organic etiology and permanent custodial placement was considered, an amytal interview[27,28] was conducted, followed by overnight DST studies. Both of these patients tended to clear in cognitive functioning during the amytal interview, and the DST revealed nonsuppression of cortisol secretion.

In my experience, the clinical dilemma of differentiating depression and pseudodementia from organic brain syndromes occurs frequently. I recommend the administration of the overnight DST early in hospitalization, usually on the second or third day, in order to obtain the DST when the patient is acutely ill and as drug-free as possible, and to expedite the diagnostic evaluation. It is desirable to diagnose and treat severe depressions as rapidly as possible because the disorder is subjectively unpleasant to the patient and carries a high rate of morbidity and mortality, especially from suicide. A speedy diagnosis tends to minimize the length of hospitalization and financial cost of treatment. Finally, the DST is easy to administer, is readily available, and is well accepted by patients. Since the DST is positive (nonsuppression of serum cortisol) in between 25-50% of cases, an abnormal DST indicates the likelihood of a major biological depression and a good response to somatic treatment.

Case Reports

The following cases illustrate the use of the DST in the diagnosis and management of primary affective illness and also its role in differential diagnosis.

Case 1

Mr. A was a 57-year-old married accountant with a long history of mood swings. He was obsessional and given to melancholic episodes, with occasional brief periods of hypomania lasting a few days and resolving without treatment. He had no history of prior psychiatric treatment. Three months prior to the index admission, he became more depressed than ever before, had anorexia with a 15-pound weight loss, and felt guilty about losing money in financial transactions, although his wife and children were supportive and loving and assured him they valued his health and well-being more than the money he lost. He declined hospitalization initially and was given doxepin 100 mg qhs and was seen in once-weekly outpatient psychotherapy, but his depression worsened and he was admitted to the psychiatric unit of a general hospital. He stared at the floor, exhibited psychomotor retardation, and expressed hypochondriacal complaints. Physical examination was normal and lab studies were negative, including the following: CBC, urinalysis, SMA-6, SMA-14, thyroid profile, chest film and EKG. However, the DST was abnormal with elevated serum cortisol levels of 19.0 mcg% and 13.5 mcg% at 4 PM and 11 PM, respectively (normal is 2.0 to 9.0 mcg%). He was given amitriptyline 250 mg qhs with gradual and marked improvement in mood. Repeat DST prior to discharge from the hospital revealed normal serum cortisol levels. The discharge diagnosis was manic-depressive illness, depressed type. He has continued to do well since hospitalization.

Case 2

Mr. B was a 71-year-old white single man with memory loss, forgetfulness, disorientation, confusion, and impaired ability to function at home. This man had always been a responsible, hard-working, inhibited gentleman whose premorbid personality was basically schizoid. Four to six months prior to admission, he began to exhibit "organicity." He took longer than usual to perform simple tasks like shaving, combing his hair, and shopping for groceries. He became confused when trying to balance his checkbook, which distressed him greatly as he had served as executor of the estates of several family members who had died in recent years and had been praised for his honesty and diligence in administering the business affairs of the family.

Admitted to a medical service in a general hospital, psychiatric consultation was requested and revealed an elderly, frail-appearing gentleman who appeared insecure, lost, quizzical and painfully aware that he wasn't the man he used to be. He could not recall the day or date and was surprisingly disoriented and confused. He denied feeling depressed but added, "I have given up." Later he told a nurse, "I know I smell bad" and had the somatic delusion that his body was stinking, a symbolic expression of his impaired cognition, physical strength, and stamina.

Physical examination was normal as were the following lab studies: CBC, urinalysis, SMA-6, SMA-14, thyroid profile, chest film and EKG. The DST revealed elevated cortisol levels of 14.4 mcg% and 10.2 mcg% at 4 PM and 11 PM, respectively. He was transferred to the psychiatric unit and given thiothixene 2 mg bid; over the next 16 days he became transformed from a senile old man into a more verbal, optimistic, and energetic man with an improved appetite for food, weight gain of eight pounds, and ability to sleep throughout the night. His improvement continued in outpatient psychotherapy and he was able to play golf again and socialize with his old friends. Clearly he was suffering from a psychotic depression that was amenable to treatment; he was a nonsuppressor on the DST. In my opinion he was fortunate to have as his family doctor a physician who was aware that not all elderly patients who appear "organic" are senile and that evaluation and treatment can sometimes achieve excellent treatment results.

Case 3

Mr. C. was a 64-year-old married businessman who was in fairly good health despite consuming a pint or more of vodka daily and for over 20 years. Despite his alcoholism he worked daily and was successful, wealthy, popular, and happy. He enjoyed the prestige of being a self-made businessman who had become successful without having any formal education and of being a civic leader with an admirable record of service to the community. Two months prior to the index admission, he received his first and only citation for driving while intoxicated, which he experienced as a narcissistic mortification. His wife and children noted an immediate change in his behavior and personality. He stopped drinking completely, lost his usual cheerfulness, was restless and discontented, unable to concentrate, worried constantly about his business and insisted he was on the verge of bankruptcy, and scoffed at the reassurance of his business associates, accountant and attorney. He would awaken at 3-4 AM and restlessly wait till he could go to work but would find excuses to stay home, in sharp contrast to his previous behavior when he would be the first man at the office. He lamented the loss of his life savings; nihilistic delusions were prominent but fortunately he was not suicidal. His family persuaded him to be admitted to a general hospital for medical evaluation; he agreed to pacify the family but insisted, "There's nothing wrong with me, I just need a lot of money for my business, that's the problem."

Psychiatric consultation led to the diagnosis of a severe depression in a man with a history of a hypomanic personality and habitual excessive drinking. He was transferred to the psychiatric unit where he was noted to have a ruddy face but otherwise normal physical examination. CBC, urinalysis, SMA-6, SMA-14, thyroid profile, chest film and EKG were normal. Psychology testing did not confirm the clinical diagnosis of a psychotic depression but instead revealed a borderline intelligence in a man with possible alcoholic brain damage and with prominent hysteroid features; anxiety and depression were in the mild range. EEG was normal and CT scan of the brain revealed mild cerebral and cerebellar cortical atrophy. The DST was normal with cortisol levels of 2.2 mcg% and 0.8 mcg% at 4 PM and 11 PM, respectively. He did quite well on a therapeutic pass and insisted on being discharged home because he felt so much better. His family reported a unique ability to "turn it on and off," bewailing his financial status (which was in reality quite good) and, upon seeing a friend or business associate, reverting to his usual extroverted affability. In view of his insomnia, depression, and obsessions, he was given amitriptyline 75 mg qhs, with immediate improvement in his sleep pattern and in

his mood and anxiety. He is the only patient I have ever examined who exhibited nihilistic delusions but was not profoundly depressed, schizophrenic, or intoxicated with alcohol or another drug of abuse. The hysterical features of his personality were considered more prominent in the clinical symptomatology than his alcoholism and documented brain damage. His normal response to the DST (DST negative) was helpful in reaching the diagnosis.

Since his symptoms were typical of a psychotic depression, he was given amitriptyline with benefit; his sleep improved and his nihilistic delusions disappeared. Yet his mood was never depressive, perhaps because of the histrionic elements in his personality. Since the DST is negative in 50% of patients with severe depression, this patient may well have had a masked depression (or depressive equivalent). Possibly the DST measures features of severe endogenous depression that reflect a biochemical heterogeneity not yet clinically understood but which future studies should clarify.

Discussion

These three cases illustrate the application of the overnight DST in the diagnosis and treatment of patients with primary affective disorder and especially in the differential diagnosis of elderly patients who present with pseudodementia, cognitive impairment, and "organicity." The return to normal of cortisol suppression of cortisol secretion after appropriate somatic treatment of depression, whether antidepressant drugs or electroconvulsive therapy, correlates with improvement in affective, cognitive, and functional performance.

Internists and family practitioners use the DST primarily to evaluate patients for possible Cushing's syndrome (hyperadrenocorticism) but may lack familiarity with its application in the evaluation of patients with affective illness. In addition to diagnostic utility, further uses of the DST include serial DSTs to monitor the course of recovery from the acute episode of depression and also to signal a possible relapse into depression should non-suppression recur during the course of recovery from depression.

Rudorfer and Clayton[21] cautioned that 50% of primary depressives may have a normal DST response, that acute medical disturbances may interfere with the test, and that two or more conditions, such as dementia and depression, may coexist. In addition, primary adrenal or pituitary disease or alcoholism may interfere with the DST.

McAllister et al[26] suggest that the DST may play an important role in the differential diagnosis of dementia and pseudodementia and suggest further research on the use of the DST in patients with true dementia or with depression complicated by severe cognitive dysfunction. Further controlled studies should prove useful in elaborating on the diagnostic validity of the DST in patients with Alzheimer's disease and in other dementing disorders.

However, Spar and Gerner[29] question whether the DST successfully differentiates dementia from depression and report early escape from cortisol suppression in demented patients without affective disease. Although most studies of the DST in depressed patients have been conducted in hospitalized patients, Amsterdam et al[2] reported early escape from suppression on the DST in some healthy outpatient control subjects and stated that many drugs and illnesses can cause spurious non-suppression. They recommended further studies with appropriate controls, including an adequate number of healthy subjects, a regard for the drug-free status of the patients, and the use of medical-surgical patients, in order to provide greater validity for the DST.

Thus, there is an impressive amount of data documenting hypothalamus-pituitary-adrenal overactivity in depression and the strategy of employing the DST to evaluate cortisol responses seems promising. At the same time, further studies are indicated to clarify and refine the clinical validity of the DST. Since only 50% of patients with severe depression will be DST-positive, another effort to improve the clinical usefulness of neuroendocrine strategies in affective disorders has been suggested by Targum et al,[17] who recommend the combined use of the DST in conjunction with the thyrotropin-releasing hormone (TRH) stimulation test, since a blunted TSH response to TRH administration has been documented in depression. They felt that the combined use of both neuroendocrine tests was justified in clinical practice. The TRH stimulation test has been helpful in identifying depressed patients but can be false-positive in the presence of primary endocrinopathy, chronic systemic disease, or alcoholism. The test is an assay of the hypothalamus-pituitary-thyroid axis. If one adds another biologic marker, the urinary 3-methoxy, 4-hydroxy phenyl glycol (MPHG), a metabolite of epinephrine,[30] there is available a growing number of neuroendocrine tests to help in the identification and treatment of patients with primary affective disease.

Finally, it may be that each test measures a poorly-understood biochemical parameter of affec-

tive illness and that each test is identifying a subgrouping of depressed patients. Further studies should prove interesting and enlightening regarding the biochemistry of these illnesses and the pharmacotherapy involved in treatment, leading to a greater understanding of these disorders.

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Was it Lyme disease?

From the
Division of Epidemiology,
Virginia State Department of Health,
with
Case Report by J. W. Kerns, MD,
Front Royal, Virginia

Case Report

A 19-year-old white male presented in June 1981 with complaint of a slightly irritated area on the back of his head of three days duration. Two days before the onset of symptoms he had removed a tick from that area. The patient's occupation involved working with tick-infested farm animals. He noticed that this tick was smaller and somewhat darker than ticks he had removed previously and was also slightly engorged.

There was no history of travel in the preceding six weeks. Examination revealed only an area 2-3 cm in diameter of warm erythematous induration of the posterior scalp and a tender posterior cervical node on the same side.

Therapy was initiated with oral doxycycline, 100 mg twice a day for the first day and once daily thereafter.

The young man returned two days later complaining of severe malaise, slight neck pain, headache,

This material was adapted from the *Epidemiology Bulletin*, the monthly newsletter of the Division of Epidemiology of the Virginia State Department of Health.

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myalgias, arthralgias and nausea and said he had felt feverish earlier that day.

On examination he appeared slightly toxic but his vital signs were normal. The original area of erythema had cleared, but expanding outward concentrically was a 6-cm ring of erythema with a purplish hue. The remainder of the exam was unremarkable, and the results of these tests were normal: complete blood count with differential, erythrocyte sedimentation rate, immunoglobulin electrophoresis, immune complex by Clq, cryoglobulins, and electrocardiogram.

The doxycycline dose was increased to 100 mg twice a day, continuing to complete a ten-day course.

The patient's systemic symptoms resolved over the next 36 hours. The skin lesion, however, increased to 8 cm in diameter by the 8th day of illness, then finally resolved by the 19th day. The patient was observed for development of cardiac, neurologic or arthritic sequelae, but in one year of followup none was noted. Sera were submitted for indirect immunofluorescence antibody testing against tick spirochete antigen, but no rise in antibody titer was detected.

Discussion

First studied in 1975 in Lyme, Connecticut, Lyme disease has since been reported in 13 other states, occurring usually in the summer months.[1] It is a systemic illness characterized by the distinctive primary skin lesion erythema chronicum migrans (ECM). Many patients have developed significant cardiac, neurologic, or arthritic complications. Such non-specific systemic symptoms as fever, chills, malaise, arthralgia and headache are usually present.

The skin lesion begins as a red macule or papule. As it expands in a circular manner, central clearing often occurs. Lesions can reach diameters of 12 inches or more, and some patients develop additional lesions. The lesions fade, lasting a median of three weeks.

Of the complications that develop in some but not all patients, the usual cardiac manifestations are atrioventricular conduction defects, and EKG changes consistent with myocarditis or pericarditis may occur. The most common neurologic manifestations are headache and stiff neck consistent with meningoencephalitis, but cranial nerve palsies and motor and sensory radiculitis may also be seen. Both cardiac and neurologic abnormalities tend to be self-limiting, although repeated episodes may occur.

The arthritic manifestations, which may begin as long as two years after the appearance of the skin lesion, are characterized by intermittent attacks of acute arthritis, usually of the large joints, with each episode lasting days to several months. About 10% of Lyme disease patients, primarily those with a history of acute arthritis, develop chronic arthritis, most commonly of the knee.

Lyme disease is thought to be caused by an infectious agent transmitted by *Ixodes* ticks. In Connecticut, about 20% of patients remember a bite by an *Ixodes dammini* tick three to 30 days prior to the appearance of ECM at the site. In California and Oregon, *I pacificus* ticks have been implicated.

Further evidence for infectious etiology lies in the success of antimicrobial therapy in altering the course of Lyme's disease. Penicillin V or tetracycline, 250 mg orally four times a day for ten days, can successfully treat the early phases of the disease, when the skin lesion is present, and can also ameliorate or perhaps prevent subsequent cardiac, neurologic and arthritic phases.

The illness described in this case report clearly matches the descriptions of erythema chronicum migrans noted in patients with Lyme disease. The serologic test did not support the diagnosis, but the antibody response may have been aborted by early antibiotic therapy. Penicillin V or tetracycline, 250 mg orally qid for ten days, can successfully treat the early phases of the disease, when the skin lesion is present, and can also ameliorate or perhaps prevent subsequent cardiac, neurologic and arthritic phases.

1. MMWR 31:367–368, 1982

Editors' Note: The suspected vectors of Lyme disease, *Ixodes dammini* ticks, recently were charged specifically with carrying a spirochete that is the causative agent of the disease.

The newly recognized spirochete was recovered in Connecticut from the blood, skin lesions (erythema chronicum migrans), and cerebrospinal fluid of three of 56 patients with Lyme disease and from 21 of 110 nymphal or adult *I dammini* ticks, according to a report from scientists at the Yale University School of Medicine, the Institutes of Health, and the Centers for Disease Control. These isolates appeared to have the same morphologic and immunologic features, and the researchers named *I dammini* as the Lyme disease culprit.[2]

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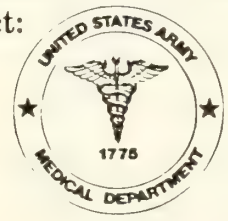

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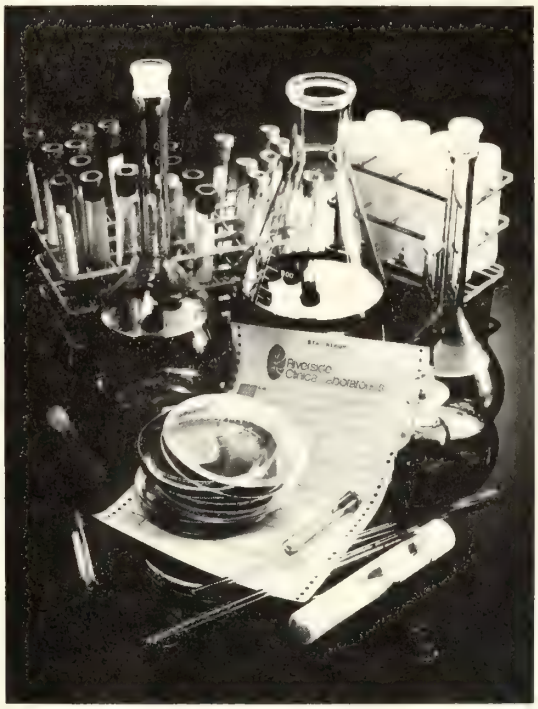

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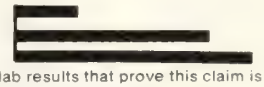

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VIRGINIA MEDICAL

EDITORIAL

Virginia's Natural Death Act: I. A "sharing of responsibility"

HIPPOCRATES did not know that medical science would develop the ability to maintain the protoplasm of the individual long after the person it contained had departed, not to return. For many people today, this ability seems to represent a threat to a basic human right which is the control and possession of one's person. Furthermore, as stated in a 1982 Joint House Resolution: "Many physicians of the Commonwealth, because of their oath to preserve life by utilizing the utmost and most extraordinary means possible, are placed in a burdensome moral dilemma, torn between the terminally ill patient's wish to die with dignity and feelings of worth, and the ability to keep such a patient clinically alive indefinitely by use of artificial-support devices. . . ." Passage by the 1983 Virginia legislature of the "Natural Death Act," as reported elsewhere in this issue, codifies the common-law right of the individual to refuse treatment and further protects the physician from liability when such expressed wishes of a patient in a terminal condition are carried out.

Was such a law desirable or necessary? Many of us, over the past seven years, during which three separate somewhat similar bills have been introduced in the Legislature, had argued against such an act. We felt that we, as physicians communicating with our patients and their families, had the situation well in hand and that legal action was not required. The public clamor persisted. Thirteen states and the District of Columbia have enacted "death with dignity" or natural death acts. Brain death acts, enacted in many states, including Virginia, proved applicable only under special and limited circumstances. "No code" orders have become commonplace; but the physician has had no legal protection even though he knew he was acting in accord with the patient's wishes and his own ethical and medical judgement. The time seemed to have come at which some better definition of the rights of the patient and of the physician in dealing with situations involving life-support systems and the terminal adult patient was required.

How did the present act happen to succeed when its predecessors had failed? The resolution passed in the 1982 session created a subcommittee for a study of the problem. As described elsewhere in this issue, the study committee included not only legislators but citizen members representing medicine, law and clergy, and the proposed bill represented a consensus of their viewpoints. A major factor was the work of Allen C. Goolsby III, counsel for The Medical Society of Virginia, who consulted with the Society's Legislative Committee.

What are some of the things the new law does? First, it gives the assurance that, by a previous declaration, an individual can participate in a decision about his or her person in a terminal situation that may render the person incapable of expressing his or her wishes at that time. It is this component that is particularly appreciated by the elderly. For example, scores of residents of Westminster-Canterbury, a Richmond retirement community,

signed a petition favoring the passage of the bill. For them, and perhaps for many of us who may someday be patients as well as physicians, the Natural Death Act can be considered as providing a measure of peace of mind. Family members, or a designated person, may make such a decision on behalf of an individual, but there are safeguards to prevent abuse. Oral statements are also acceptable if made in the presence of a physician and two witnesses, but the fact of such a declaration should be documented in the medical record.

Secondly, the Act provides vey broad indemnification to the physician who honors a patient's wishes in such a situation. This may be considered as providing some peace of mind to the physician, who thereby knows that he cannot be subjected to criminal prosecution or civil liability, or be deemed to have engaged in unprofessional conduct as a result of the withholding or withdrawing of life-prolonging procedures from a patient with a terminal condition.

There are some things the Natural Death Act does not do. It provides no committees or other mechanisms for deciding that a terminal condition exists in a particular case; that remains, rightly, a medical decision, and its definition is not always easy. As defined in the Act: "Terminal condition means a condition caused by injury, disease or illness from which, to a reasonable degree of medical certainty, (i) there can be no recovery and (ii) death is imminent." The Act applies only to cases in which this "terminal condition" has already been recognized to exist. In most cases, such a determination will, obviously, be shared by more than one physician, but this is not specified. The thrust of the measure is that of defining an appropriate course of action in previously designated terminal cases in which the patient has declared his wish not to have his dying prolonged. Furthermore, the Act does not require that a physician comply with the declaration of a patient or his appropriate designee but does specify that, if the physician is unwilling to do so, a reasonable effort to transfer the patient to another physician should be made. It is probable that, in most cases, a physician's refusal would be based on his judgement that a "terminal condition" does not yet exist; and, if it indeed does not, the provisions of this Act are inapplicable.

As physicians, we generally believe in "fighting to the finish" for meaningful life for our patients. That very respect for human life precludes "fighting *beyond* the finish." Virginia's Natural Death Act can prevent concern over our being legally required to do so and provides for the appropriate sharing of responsibility by patient and physician.

SUSAN J. MELLETTE, MD

Box 207, MCV Station
Richmond VA 23298

II. "The dying need no passports"

VIRGINIA is now the fifteenth state to have legislated a "right-to-die" law that gives legal strength to "living wills" to end life-sustaining measures in the event of terminal disease. These efforts to control terminal care relate to the increasing number of patients inordinately sustained on positive pressure respirators and the entire paraphenalia of intensive care unit support systems. Life-sustaining technology has dramatically improved, as has the team structure that maintains it and the insurance and federal support that pays for it. There is no question that patients are now inappropriately kept alive and that some measures are necessary to help the patient, family, physicians and state to make decisions related to ending life where there is loss of quality existence and conscious awareness.

The problem with "right-to-die" laws that give legal strength to ending life in the event of terminal disease is that they are applicable to only a small minority of patients. Few patients will provide living wills and still fewer will be helped by this legal document in their possession. It is my contention that the issue of life termination has to be faced in the setting where heroic measures really begin, i.e., in the intensive care unit.

It is in the setting of the ICU that evaluation and prognosis can be made and where physicians and families can control the problem.

Here are some case histories to illustrate my point.

First, my 82-year-old widowed aunt, with no surviving children, recently suffered her third major myocardial infarction and shortly afterwards developed intractable pulmonary edema. Her closest friend and relative was called by the cardiologist, who suggested that the only way to sustain the patient was to put her on a positive-pressure respi-

rator. There was little hope, he said, but a "miracle" might occur. This produced the consent. The tracheotomy was done, the respirator applied, and the patient maintained in the ICU for four weeks, during which time she was semi-comatose and hardly able to communicate with friends and family. Finally, when the ICU bed was needed for a patient with a more hopeful prognosis, she was moved to a private room where, still on a respirator, she suffered the final indignity of dying in a hypothermic blanket, on antibiotics for an overwhelming infection which, mercifully, killed her.

Nowhere did the patient's physician sit down with the family to inform them that there was no hope for this lovely lady and that it would be best for the patient and those participating in the anguish of her suffering that the patient be sedated and allowed to die.

Case 2: An 84-year-old woman, with a feeding tube, in coma for four years, is admitted to a teaching hospital by her family doctor for the sixth time with pneumonia. Her skin is in perfect condition; she is the devoted focus of her family's attention. The housestaff, with the aid of fluids, oxygen and antibiotics rescues her, to go home again.

No one discusses with this family the ethical and medical issues of the patient's survival and repeated hospitalization. There is no vehicle in the hospital, during repeated admissions, for discussion of the issue of heroic care with those truly responsible for the patient.

Case 3: A 76-year-old man with bladder carcinoma metastatic to lung and aortic nodes is admitted with seizure followed by hemiparesis and coma. He is suffering from renal failure, respiratory insufficiency, possible metastatic disease to the brain, and EKG evidence of a myocardial infarction. The attending urges the housestaff to stop sustaining the patient, but primary care responsibility is subjugated to a learning experience about water, electrolyte balance and the control of infection and respiratory gases. The patient is maintained in perfect balance for three weeks. Then, without a word to the family, the patient is moved off the ICU and, on a less efficient respirator and without the care of the ICU team, dies within 24 hours.

Throughout the period of intensive care, the family is fed the mythology of the hopeful miracle, but is never given a participatory role relevant to the prognosis.

Let us be candid. The intensive care unit's appropriate role is to keep the patient going on a day-to-day basis—to sustain life, not judge its quality or cost.

THE PROBLEM of life withdrawal will not be solved by a living will sitting in a patient's back pocket or in a lawyer's vault.

We need truly valid informed consent for families related to the prognosis and cost of life support. We need to help families accept death and to provide them with adequate information as to what the machinery of life support is all about. We need the courage of our convictions that to keep a respirator going can be wrong, and that if our peers accept our position and this has been openly discussed, we can stop the machine.

As physicians, we need to reserve the right to withdraw from a case if a family insists, in an irrational manner, that we must continue to use life support when it is against our convictions. This does not demean our responsibility to support life, for it is not our responsibility to pander to a mechanically driven corpse.

Our hospitals, most particularly those with intensive care units, need to create "ethical care committees," whose members, at the family's or physician's request, will sit down with the family and others primarily responsible for the terminally ill patient to discuss the patient's course and prognosis and the relevant use of heroic measures. I feel that our fears of medical malpractice or murder charges will not be valid if the exchange of information via such an ethical care committee is available, with a written document attesting to prognosis.

We should not be afraid of our traditional responsibility for life or death, provided that we have the backing of our peers, made visible through open communication. Although lawyers outnumber us three to one, the courts should be supportive if we make visible the process of decision-making that involves those concerned.

We do not need expensive legal symbols and the dying do not need passports. What is required is a responsible, formalized method of informational exchange that allows us to accomplish our primary duties, which are, first, to support life, and second, when all else fails, to be kind.

WILLIAM REGELSON, MD

Box 273, MCV Station
Richmond VA 23298

VIRGINIA MEDICAL OBITUARY

Alvin C. Wyman, MD

Dr. Alvin C. Wyman, Washington, DC, died following a heart attack on March 28 at the George Washington University Medical Center, where he was clinical professor of radiology. He was 64 years old.

Born in New York City, Dr. Wyman was graduated from Cornell University and received his medical degree from the New York University College of Medicine in 1943, then trained at the Naval Hospital in Norfolk. He served as a lieutenant in the United States Navy Medical Corps from 1943 to 1947 and for the following ten years was a lieutenant in the Naval Reserve.

Dr. Wyman had served as chief of the Medical Center's division of diagnostic radiology, had been chief of radiology at Alexandria Hospital, and had served for 35 years as a radiologist for the Alexandria Department of Health. He was a consultant in radiology at the Children's Hospital National Medical Center and the US Naval Hospital in Bethesda.

A member of The Medical Society of Virginia and the Alexandria Medical Society since 1947, Dr. Wyman also belonged to many other professional organizations, including the American Roentgen Ray Society, the American College of Radiology, the Radiological Society of North America, and the American College of Physicians.

Reuben F. Simms, MD

Dr. Reuben Foster Simms, general practitioner in Richmond for more than 50 years, died the night of April 26 in a Richmond hospital. He was 80 years old and on the day of his death had seen patients for two hours in his office and then made a house call.

A native of West Point, Virginia, Dr. Simms was graduated from the College of William and Mary, went on to the Medical College of Virginia, graduating in the Class of 1928, and then trained at Richmond's Stuart Circle Hospital. He had been a district governor of Lions Club International, with whose program on behalf of the blind he was actively involved.

Dr. Simms had belonged to The Medical Society of Virginia and the Richmond Academy of Medicine for 52 years and held memberships also in the Southern and American Medical Associations and the American Academy of Family Physicians.

James W. Todd, MD

Dr. James Wallace Todd, a surgeon in Virginia Beach for nearly 20 years before moving to Florida in 1962, died at his home in Eustis, Florida, on March 3. He was 67 years old.

A native of Belton, South Carolina, Dr. Todd was graduated in 1940 from Emory University, Atlanta, and received training in Norfolk at Norfolk General and De Paul Hospitals. During World War II, from 1943 to 1945, he served in the military. Three years later, working with the late Dr. H. Floyd Dormire and Dr. Waller L. Taylor, Sr., Dr. Todd helped found Virginia Beach General Hospital.

Dr. Todd joined The Medical Society of Virginia in 1947 and retained his membership throughout his Florida residence. In Florida, he was elected to the presidency of the Lake County Medical Society. He belonged also to the American College of Surgeons and the Southeastern Surgical Congress.

T. G. Boisclair, MD

Dr. Thomas George Boisclair, Richmond psychiatrist, died April 18 at his home at the age of 62. He was a graduate of the George Washington University School of Medicine and trained at the Medical College of Virginia.

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WHO'S WHO

All by himself, **Dr. Harry J. Warthen** is a multidisciplinary team of surgeon, historian, editor and curator. And in each of these specialties he has attained prodigious tenure and many honors, as follows:

For 38 years esteemed physician and good citizen of Richmond. President of the Richmond Academy of Medicine. Recipient of The Medical Society of Virginia's Physician Award for Community Service. Chairman from 1955-1960 of Richmond's Board of Health. A founder and first chief of surgery of Richmond Memorial Hospital.

Historian and Civil War buff for a long, long time, some say since his childhood in Alexandria, where the Warthen family home was next door to a house in which Robert E. Lee had lived. President of the Antiquarian Society of Richmond. Trustee of the Historic Richmond Foundation. Member of the Confederate Round Table and the Sons of Confederate Veterans.

For 20 years the scholarly Editor of the VIRGINIA MEDICAL MONTHLY, racking up a total of 206 editorials and myriad historical chronicles both medical and Virginian. Now the journal's Editor Emeritus. Originator of The Medical Society of Virginia's official seal. President of the University of Virginia's Medical Alumni Association.

Curator of the Richmond Academy of Medicine's archives from 1957 until January of this year, during which quarter of a century he not only saw to the care and feeding of the academy's fine collection of early books on medicine but mounted three major exhibits: one commemorating the 250th anniversary of the founding of Jamestown, another extensively encompassing Civil War medicine, and a third on medicine of the Revolutionary War period in celebration of the 1976 Bicentennial year.

In turn, the Richmond Academy mounted a salute to Dr. Warthen, complete with plaque and encomium. These were presented at a spring meeting of the academy by the honoree's long-time friend, **Dr. John P. Lynch.**

Referring to Dr. Warthen's accomplishments as "awesome," Dr. Lynch made the long trip through the 81-year-old surgeon's CV and then summed up: "He has honored us with his distinguished career and contributions to the community and this academy, and these will live as long as there is a Virginia."

The chairman of MCV's Department of Pathology, **Dr. George P. Vennart,** has been appointed Southeast Regional Commissioner for the College of American Pathologists' Commission on Laboratory Accreditation.

Dr. James H. Bowden, Abingdon, has been named president of the newly formed Washington County Virginia Unit of the American Heart Association.

In testimony to **Dr. Thomas McPherson Brown,** Arlington, patients and associates from all over the country joined in a dinner at Washington's Mayflower Hotel. Widely known for his pioneering work in rheumatology, Dr. Brown is director of the Arthritis Institute, Bethesda, Maryland.

Dr. Walter E. Nance, Richmond, has been elected to the board of directors of the American Board of Medical Genetics. He is chairman of the Department of Medical Genetics at the Medical College of Virginia.

For a multidisciplinary team, another honor: Dr. Warthen.

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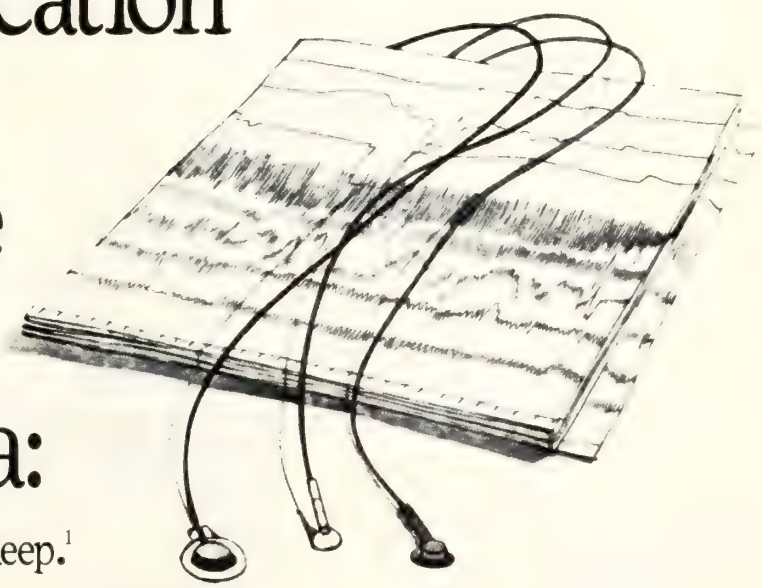
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From the VAMEDNE

Exploring the options: a union? an IPA?

SMARTING from assaults on the traditional ways of practicing medicine, members of the Richmond Academy of Medicine met in special session on the last day of May to hear advocates of two ways of fighting back.

It was the first such called meeting in 16 years, and it followed by three weeks a regular meeting at which Dr. Richardson Grinnan had described Blue Cross Blue Shield of Virginia's new preferred provider organization.[1] (Oddly enough, the special session in 1967 also was triggered by the announcement of a BC/BS innovation—the now-familiar usual, customary and reasonable fee schedule.)

Dr. Davis

The immediate sequela to Dr. Grinnan's presentation was, according to one long-time member, "the most emotional Academy meeting I have ever seen." In answer to the commotion, Dr. Ronald K. Davis, Academy president, appointed Dr. Emerson D. Farley, Jr., to head a task force to look into "competitive alternatives," not just to PPOs but to all the other recent incursions into medical territory.

From the task force's deliberations sprang the extra meeting and its two speakers. An estimated 375 members turned out to listen. At the outset, Dr. Davis told them that no resolutions would be entertained; the meeting, he cautioned, was for informational purposes only.

Dr. Farley brought on the first speaker, Michael E. Herbert, president since 1976 of Physicians Health Services in Bridgeport, Connecticut. At age 38, Herbert administers an IPA-HMO that has as participants 550 physicians, eight hospitals and 27,000 enrollees. As is typical, the HMO is the administrative arm; the IPA is a separate legal entity through which the physicians contract with the HMO to provide services to the enrollees while continuing to practice on a fee-for-service basis in their usual solo, group or partnership settings.[2]

The fee schedule approximates the charges of physicians in the area, Herbert said, and the participating physicians are paid 80% of that schedule at the time of service; the other 20% is set aside in a "risk pool." If at the end of the year the books are in the black, the pool is distributed to the physicians.

By and large, Herbert recounted, the association's financial history has been decidedly healthy; an episode of fiscal peril not long ago was licked by instituting such strictures as pre-certifying hospital admissions, auditing hospital bills, and sanctioning some doctors whose costs were out of line. All these controls, Herbert admitted, should have been in place from the outset.

"An IPA-HMO offers you a way to be organized," Herbert concluded, "and you must be organized because the competition is organized." Observing that "This is an innovative society," he predicted "new challenges we haven't even thought of yet."

For those thinking of forming an IPA-HMO, Herbert recommended 1) $1 million in capital; 2) a willingness on the part of the participating

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VSB BUREAU

Doctoring at the Summit: heady duty

RIGHT smack in the middle of the Summit Conference heard 'round the world were two dozen Medical Society of Virginia members. They were on rotating duty at three stations set up to cope with medical emergencies in the crowd of 9,000 people that jammed Williamsburg for the meeting.

Experts had predicted at least two heart attacks in such a throng; these did not occur, happily, although full-dress resuscitative equipment was at the ready, but in the heady atmosphere of international diplomacy the minor illnesses and injuries that did present became memorable events.

The White House and the State Department had asked The Medical Society of Virginia to set up the stations, lock, stock and staffing. It sounded like a tall order, but Dr. Harold L. Williams, MSV President, and the Council accepted the invitation forthwith.

The search was on for the members who would staff the stations. Orientation to emergency medicine was a criterion, so was facility in foreign tongues, so was availability over a four-day span. Thirteen nurses from the Williamsburg/Newport News area were lined up to assist the physicians.

Dr. Williams undertook the formidable task of furnishing the stations and moving the equipment and supplies into place. Thanks largely to Riverside Hospital and the Fire Department in Newport News, which loaned or donated everything from aspirin to defribillators, the stations were models of their kind. They were located at the Williamsburg Inn and the Lodge, where the delegates came and went, and at William and Mary Hall, where the press was headquartered.

These press people generated most of the medical action, suffering lots of cuts and abrasions while "dragging all that heavy photographic equipment around," as Dr. Reinald Leidelmeyer of Fairfax put it. Dr. Leidelmeyer speaks not only English but German, Dutch and French; the last came in handy when a French cameraman came in with a cut on his nose. He also treated a case of shingles and had his share of sunburn cases.

A member of the Japanese delegation came in for treatment of a nasty sty on his eyelid and was greeted in his native language by Dr. Junichiro L. Nakanishi of Virginia Beach, who accompanied the afflicted man to the Williamsburg Community Hospital for surgery. Also to the hospital went a kitchen worker who fell and bruised his head during what appeared to be an epileptic seizure.

Complaints of severe headache were frequent, largely because, in the observation of Dr. Frederick K. McCune of Virginia Beach, "these people are working under tremendous pressure and seem to be getting very little sleep. One man who

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Stewart MacInnis

Dr. Williams, left, and Dr. McCune; at rear, Margaret Nelson, RN

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Options

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physicians to change practice patterns; 3) careful management.

There was exploration of IPA feasibility in Northern Virginia in 1980,[2] but the project failed of consensus among physicians of the area. In Norfolk there have been reports of tentative movements toward an IPA for several years, but those seem now to be quiescent.

The second speaker was introduced by Dr. I. Norman Sporn. He was Stanley S. Peterson, MD, Springfield, Missouri, internist, and he has been president of the American Federation of Physicians and Dentists since its formation ten years ago, when it welded together a group of small, union-type organizations scattered over the country. It now has chapters in 38 states and a membership of 18,000.

Tall and white-haired, the patriarchal Dr. Peterson early on handed down to his assembled peers a dictum to live by. "I will remind you," he said, giving each word weight, "that physicians are the only physicians in business."

Getting into the subject of union membership, he chided his audience in a way no outsider would dare. "This may come as a shock to you, but nobody gives a damn about your image," he said. "Your patients want you to treat their problems and do a good job of it. That's all they care about."

The Federation has never had a strike, he pointed out, and it is not affiliated with the Teamsters, the AFL-CIO, or such like. "They don't want us, we're too bothersome," he said with an expression of mock disdain that drew laughs from the crowd.

His recommendations for conducting a Federation chapter, however, were altogether serious. On staff: "Get yourself a good labor lawyer who is solidly on your side, someone who is a little venturesome." On the Federal Trade Commission: "State law supercedes the FTC. Look at your state law." On lobbying: "See to it that the lawyers who dominate your legislature understand that *they're next.*"

And finally, "Be optimistic," said this man of great faith who for ten years has been going up against what appears to be a deep-rooted physician resistance to unionization.

Questions from the floor then were honored by Dr. Davis. One Academy member came to the microphone to ask, "I think of a union's purpose as dealing with management, but I'm self-employed, I'm my own manager, so what's the point?"

To negotiate with governmental agencies, insurance companies, HMOs and DRGs and PPOs and all the other schemes that are moving in, Dr. Peterson responded.

"Isn't it possible," another physician asked rhetorically, "that we don't need a union or an IPA but should just let the great leadership of our Academy and our state society negotiate for us? I don't see why we have to get so fancy."

"I'm not against organized medicine," Dr. Peterson told Virginia Medical's reporter. "If we didn't have organized medicine we'd be in a helluva shape. That would be the best way to work, through the local and state societies, but they just aren't put together so that they can do it. We need something more." Dr. Peterson was for 14 years an AMA delegate and is now an alternate delegate from the Missouri State Medical Association.

A few days after the special meeting, a dozen doctors who had been in the audience met to consid-

Summit

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came in with a headache simply needed to lie down and go to sleep."

Television cameramen devoted a lot of footage to the physicians. Reporters both foreign and domestic asked a lot of questions and filed a lot of stories. One of the White House physicians stopped by the medical station at the Inn. So did Virginia's two senators, John Warner and Paul Trible.

At noon on Tuesday, as the Reagans were to board a helicopter to return to Washington, all those who had staffed and secured and fed and otherwise served the conference gathered on the lawn behind the Williamsburg Inn, there to hear the President thank them for their hard work. Then he and his wife waved good-by, boarded the aircraft, and away they went. It was "a very moving occasion," Dr. Williams recalls. "It gave us a real sense of history. I'm really proud of those physicians and nurses and everyone who helped us. It was a big job, but we'd be glad to do it all over again."

Helping Dr. Williams with supervisory responsibilities at the Summit were Dr. C. Barrie Cook, MSV President Elect, and Dr. Armistead Williams, medical director, Colonial Williamsburg Foundation. And helping coordinate the effort was C. E. Douglas, VaMPAC staff man who free-lances on occasion for the State Department and was Deputy Director of Operations for the Summit.

In addition to Dr. Leidelmeyer, Dr. Nakanishi and Dr. McCune, these physicians contributed tours of duty at the Summit stations: Dr. Harry C. Kuykendall, Alexandria; Dr. Samuel D. Caughron, Charlottesville; Dr. Branko Sergio Valenti and Dr. Yasauki Takagi, Fairfax; Dr. Gaylord W. Ray, Gloucester; Dr. Fumikazu Kawakami, Hopewell; Dr. Webb D. Jones, Dr. Walter P. Lowery, Dr. David Muffelman, Dr. Rudolph J. Naurath and Dr. Ronald J. Rejzer, Newport News; Dr. Russell D. Evett, Dr. Daniel H. Kinzie IV and Dr. Vernon L. Cofer, Jr., Norfolk; Dr. William L. Harrell, Staunton; Dr. Louis Farano III, Virginia Beach; Dr. Jon Robert Hammersburg, Waynesboro; Dr. James B. Hess and Dr. Peter A. Sim, Williamsburg.

According to a Colonial Williamsburg spokesman, the volunteer physicians helped not only the conference but its landmark setting. The meeting produced, he said, "absolutely fantastic international publicity" and a record number of donations. —ANN GRAY

---

er the formation of a Federation chapter in Virginia. They elected Dr. Sporn as chairman and decided to invite all physicians in Central Virginia to a meeting so that broad sentiment for a union chapter could be assessed.

As for the IPA idea, Dr. Farley predicted that "based on the interest shown at this meeting," the Task Force would ask the Academy to evaluate the feasibility of establishing alternative delivery systems in Virginia. He specified three criteria: 1) control by physicians; 2) ability to compete; 3) maintenance of "the traditional values of private practice most of us hold so dear."

—ANN GRAY

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2. Gray A: Virginia Medical's guide to HMO-watching. Va Med 107:764-771, 1980

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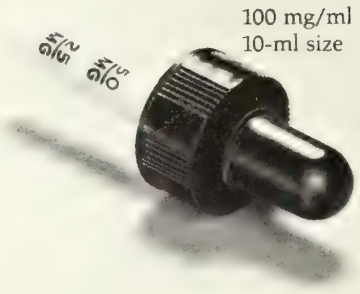

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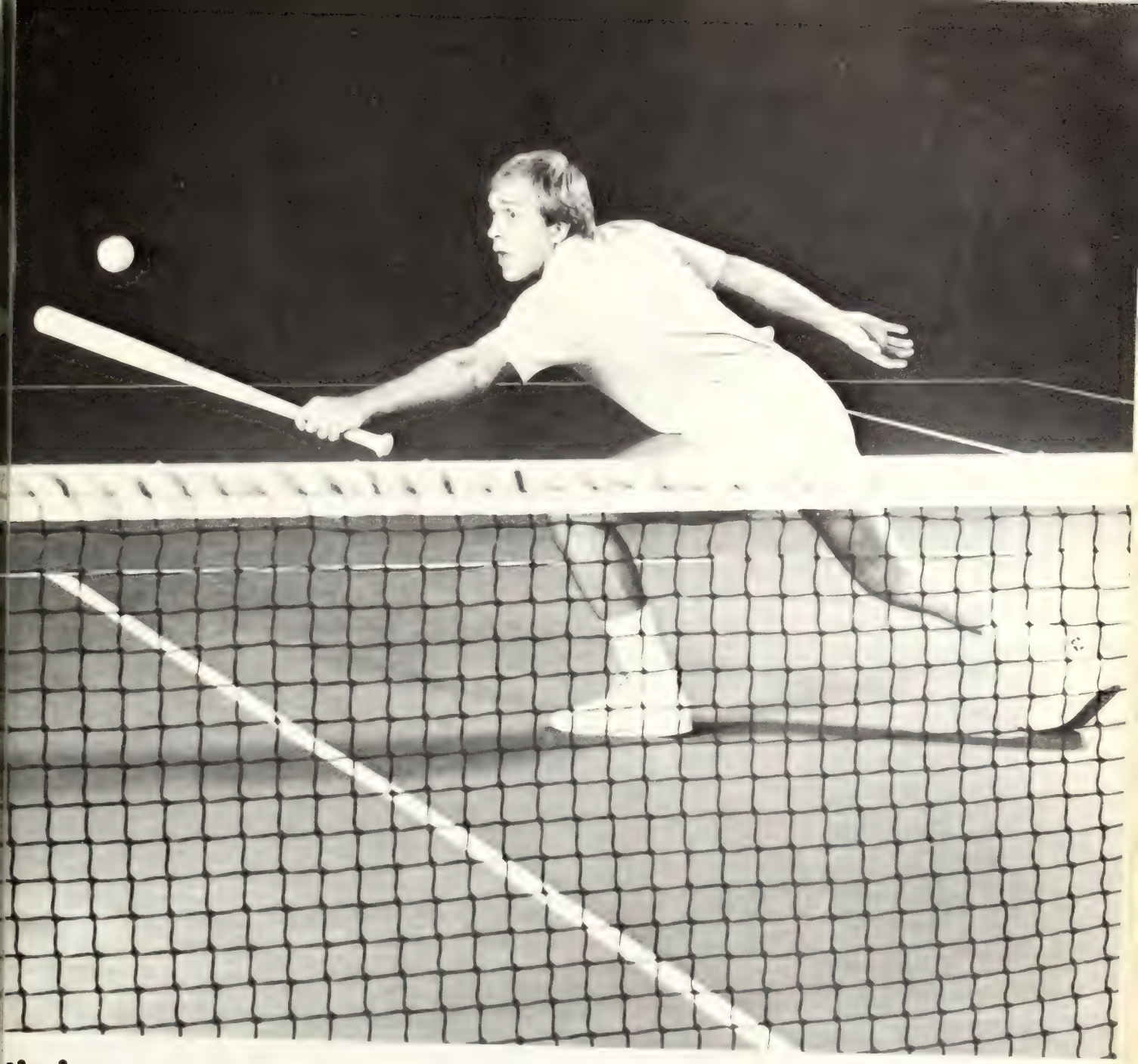

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Melissa Grimes-Guy

To Washington

went Medical Society of Virginia officers and councilors on a May day, to be joined there by Virginia's legislators for the Society's annual congressional luncheon. As in the past, they gathered together on the Senate side of the Capitol in the Mike Mansfield Room, the small mahagony-panelled dining room dominated by a portrait of the longtime senate majority leader who is now Ambassador to Japan.

Earlier there had been a tour of the White House, from whence the group went to the Department of Health and Human Services. There, in a presentation peppered with such acronyms as PPO and DRG, a spokesman described the Reagan administration's plan to cut health-care costs by reorganizing the system on a free-market basis.

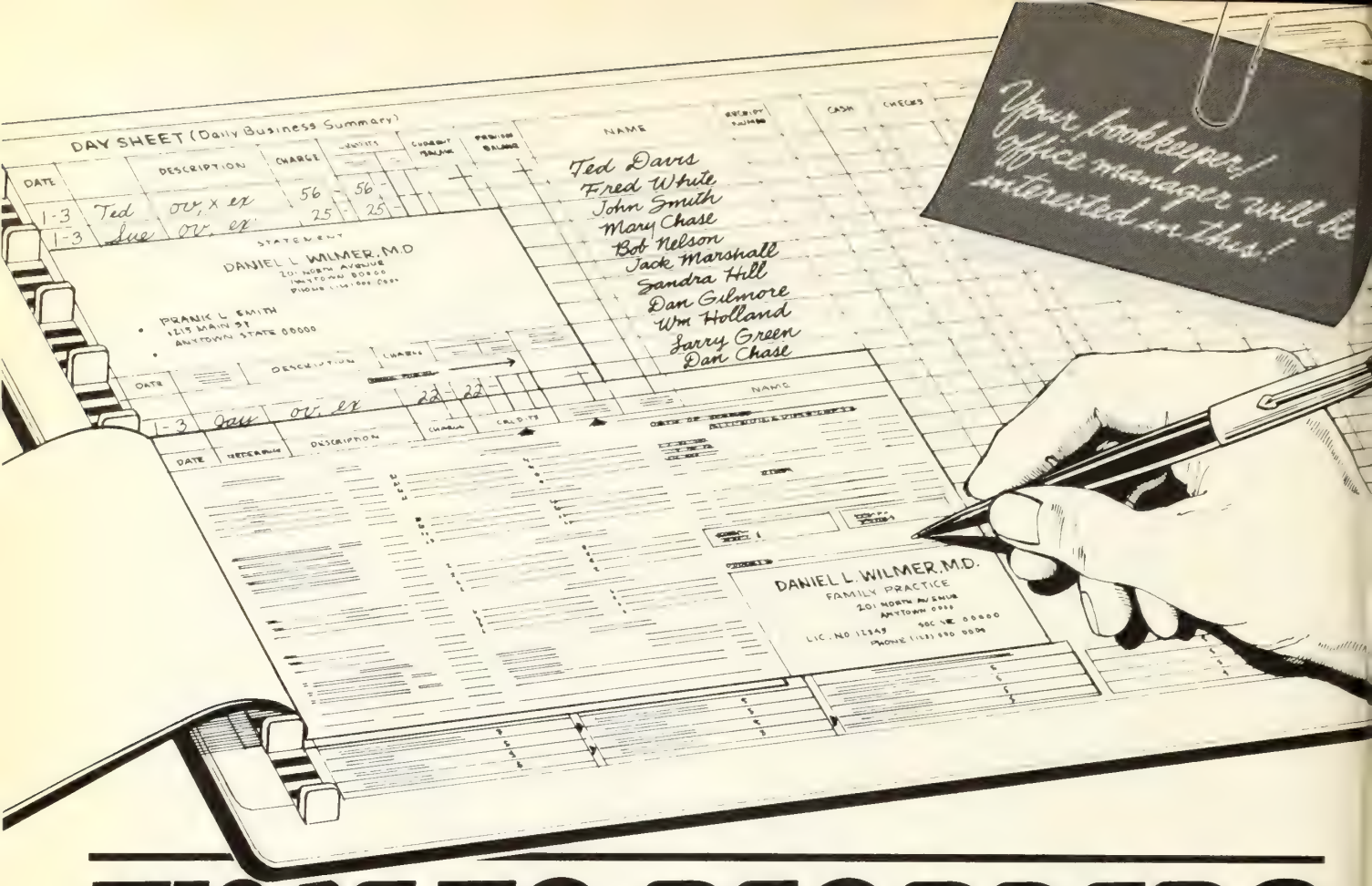
▲ Dr. F. Ashton Carmines, left, and Rep. H. H. Bateman (R-1st district)

▲ Rep. Norman Sisisky (D-4th district), left, and Dr. H. Alan Bigley

▼ Dr. C. Barrie Cook, left, and Sen. John W. Warner

▲ Frank R. Wolf (R-10th district), left, and Dr. Donald S. Thorn

◄ Opposite page: Dr. William W. D. Butler III stands to introduce Rep. James R. Olin (D-6th district). Above them hangs Mike Mansfield's portrait.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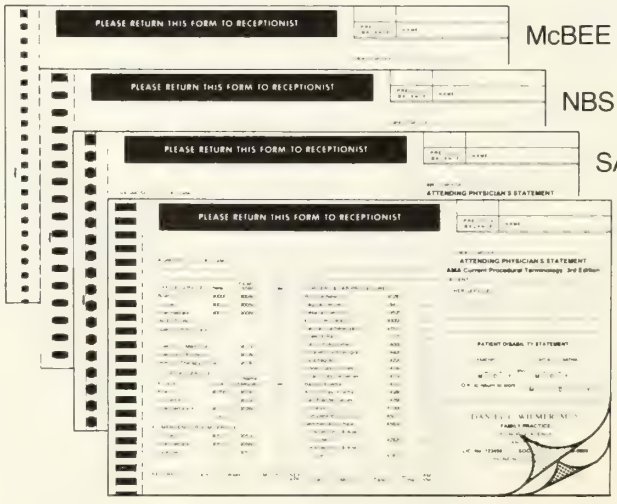
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MEETINGS ABOUT MEDICINE

July 22-24
Advances in Cardiology (Medical College of Virginia), *Hot Springs*. Mrs. Randy Casey, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

July 28-31
Practical Office Medicine (Virginia and North Carolina Societies of Internal Medicine and Eastern Virginia Medical School), *Virginia Beach*. 12 credit hrs. Fee: $50 per day or $135 for entire meeting. Virginia Society of Internal Medicine, PO Box 6264, Norfolk VA 23508, (804) 446-5243.

July 28-30
5th Annual Pediatric Primary Care Conference (Medical College of Virginia), *Virginia Beach*. 12¾ Cat. I credits. Fee: $250. Sheri Rosner, Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

July 28-30
Internal Medicine Summer Seminar (Eastern Virginia Medical School), *Virginia Beach*. James P. Baker, MD, EVMS, PO Box 1980, Norfolk VA 23510.

August 1-6
Sports and Tennis Medicine Symposium (Virginia Sports Medicine and Rehabilitation Institute), *Sweetbriar College*. Cat. I credits. Robert P. Nirschl, MD, 3801 N. Fairfax Dr., Arlington VA 22203, (703) 525-5542.

August 10-13
Summer Retreat: Practical Issues in Primary Care (Medical College of Virginia), *Virginia Beach*. 17½ Cat. I credits. Fee: $235. Office of CME, Box 48, MCV Station, Richmond VA 23298. (804) 786-0494.

August 11-13
Practice Management for the Emergency Physician (Virginia Chapter, American College of Emergency Physicians), Cavalier Hotel, *Virginia Beach*. 22 hrs Cat 1. Fee: $125-$400. Gwen E. Messler, PO Box 317, Highland Springs VA 23075, (804) 737-9433.

August 18-20
Practical Dermatology for the Primary Care Physician (Eastern Virginia Medical School), *Virginia Beach*. 14 credit hrs. Fee: $175. Office of CME, EVMS, PO Box 1980, Norfolk VA 23501.

August 18-21
Common Concerns of Plastic and General Surgeons (Eastern Virginia Medical School), *Virginia Beach*. Office of CME, EVMS, PO Box 1980, Norfolk VA 23501.

August 25-27
Reconstruction of the Breast (Medical College of Virginia), *Hot Springs*. 14½ credit hrs. Fee: $350. Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

August 25-27
Annual Meeting, West Virginia State Medical Association, *White Sulphur Springs, West Virginia*. C. R. Lewis, PO Box 1031, Charleston WV 25324.

August 26-28
Medical Malpractice Seminar (Southern Medical Association), *Hot Springs*. Fee: $220 for SMA members, $275 for non-members. Jeanette Stone, SMA, PO Box 2446, Birmingham AL 35201, (205) 323-4400.

September 7
Hypertension and Women (Eastern Virginia Medical School), *Hampton*. 4 credit hrs. Fee: $40. Office of CME, PO Box 1980, Norfolk VA 23501, (804) 446-5240.

September 11
1st Annual Citizens Trust Lecture (Eastern Virginia Medical School), *Portsmouth*. Lou Ann Hamer, Office of CME, PO Box 1980, Norfolk VA 23501, (804) 446-5240.

September 11-15
Clinical Skills Workshop and Review (Bowman Gray School of Medicine), *Winston-Salem, North Carolina*. 31 hrs., Cat I. Fee: $360. Division of CME, 300 Hawthorne Rd., Winston-Salem NC 27103.

September 15-16
Advances in Diagnostic Ultrasound (Johns Hopkins University), *Baltimore*. 16 credit hrs. Julia C. Burbridge, Office of CME, 720 Rutland Ave., Baltimore MD 21201, (301) 955-8450.

September 15
Anti-coagulant, Thrombolytic and Anti-platelet Therapy (Roanoke Memorial Hospital), *Roanoke*. Charles L.

Crockett, MD, Roanoke Memorial Hospital, Roanoke VA 24033, (703) 981-7228.

September 19-21
National Symposium on Colorectal Tumors (George Washington University), *Washington, DC*. Cat I credits. Fee. $395. Greg P. Thomas, Office of CME, 2300 K St., NW, Washington DC 20037, (202) 676-4285.

September 25-28
Radiology Update (University of Virginia), *Charlottesville*. 20 credit hrs. Fee: $350. T. E. Keats, MD, Box 170, UVa Medical Center, Charlottesville VA 22908, (804) 924-2781.

September 26-30
Basic Practices for the Novice Ultrasonographer (Johns Hopkins University), *Baltimore*. 40 credit hrs. Fee: $400. Office of CME, 720 Rutland Ave., Baltimore MD 21201, (301) 955-8450.

September 27-29
Treatment of Hyperglyceridemia (National Institutes of Health), *Bethesda, Maryland*. Mrs. Peter Murphy, 2115 E. Jefferson St., Suite 401, Rockville MD 20852, (301) 468-6555.

September 30-October 2
Internal Medicine Symposium (Eastern Virginia Medical School), *Virginia Beach*. Office of CME, PO Box 1980, Norfolk VA 23501, (804) 446-5240.

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References
1. Stone PH, Turi ZG, Muller JE. Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104 672-681, September 1982
2. Antman E, Muller J, Goldberg S, et al. Nifedipine therapy for coronary-artery spasm: Experience in 127 patients. *N Engl J Med* 302 1269-1273, June 5, 1980

BRIEF SUMMARY
PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug Interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request

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While this patient's experience
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3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

Please see PROCARDIA *brief summary on adjoining page*

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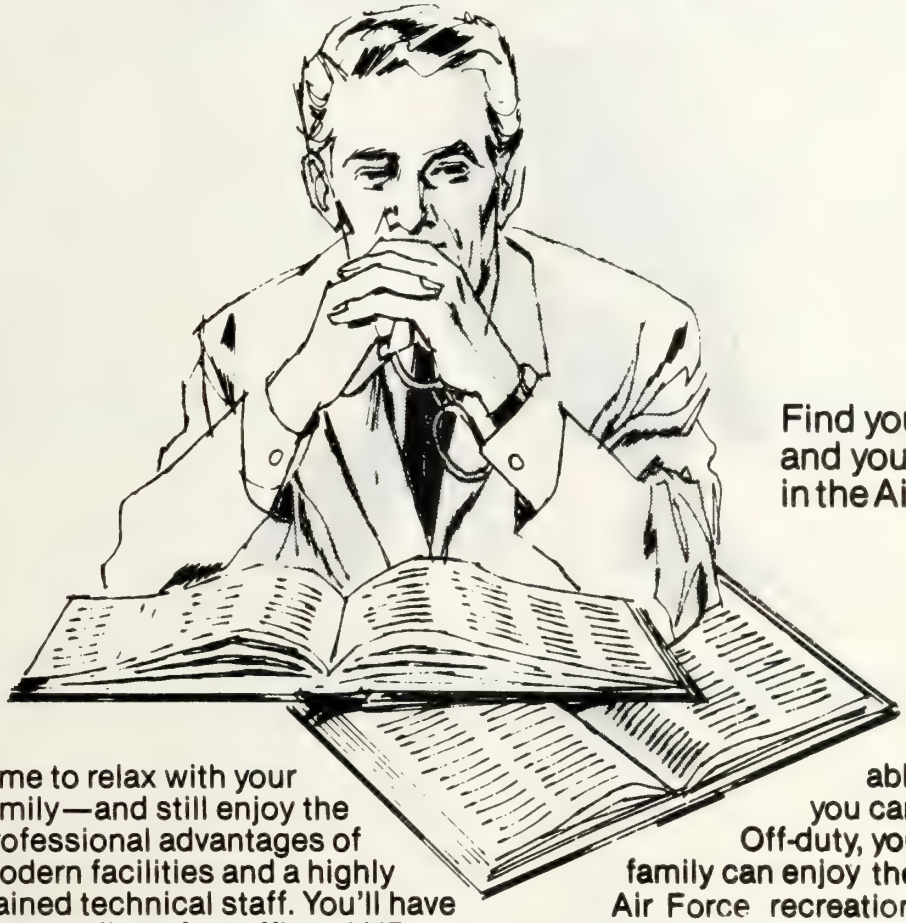

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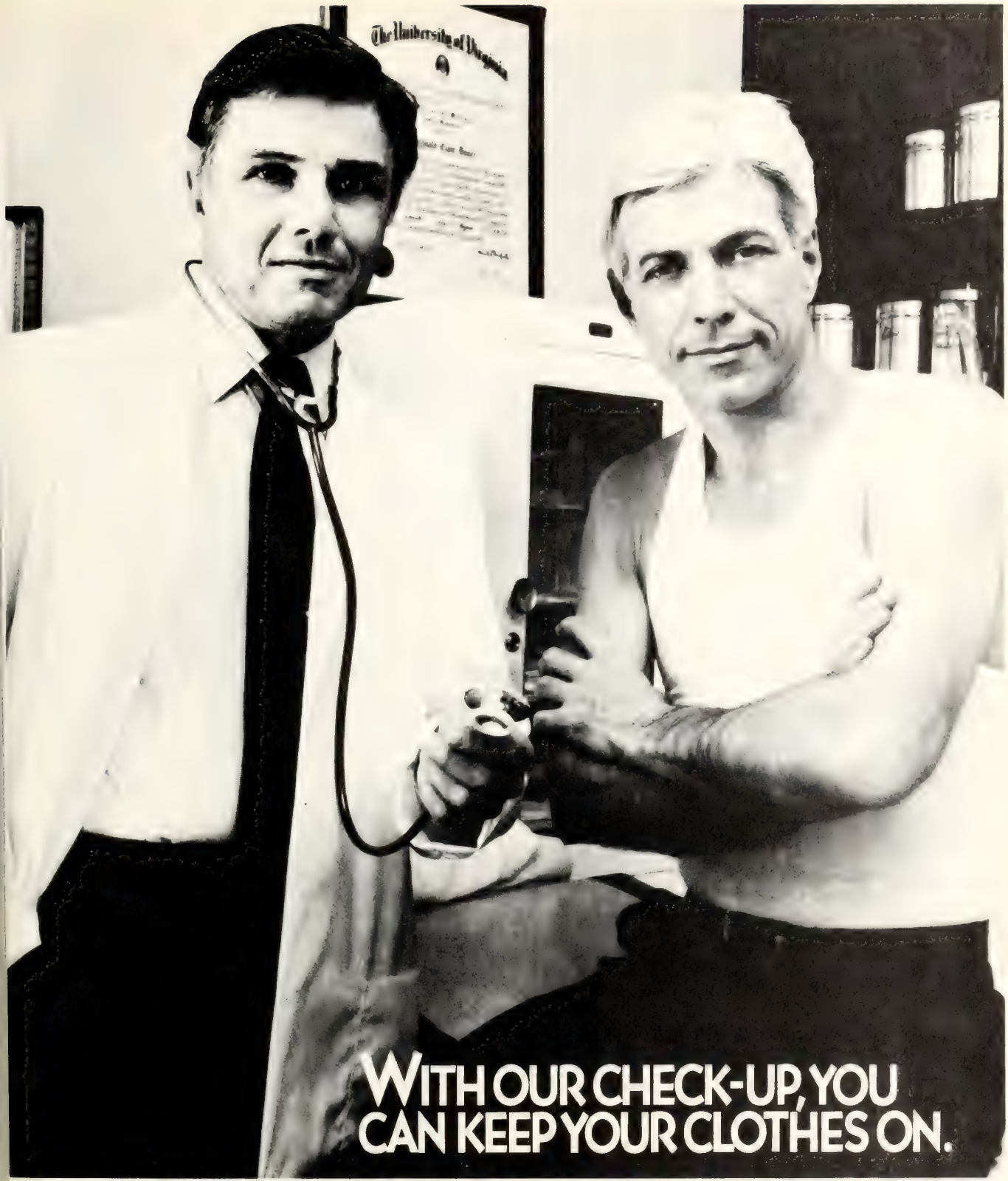

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Connective Tissue Diseases and the Cardiovascular System: a Review

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Cardiovascular problems accompany many connective tissue diseases. Rheumatoid arthritis, for example, carries a high incidence of associated cardiovascular pathology, while in Behcet's disease the incidence is low. Among these secondary abnormalities, the most frequently documented by the author are pericarditis, diseases of the aorta and myocardium, and vasculitis.

MANY of the connective tissue, or rheumatic, diseases affect the cardiovascular system. The frequency of involvement ranges from 3%-30% in all patients with connective tissue diseases. The clinical presentations vary; the patient may present with an acute and severe cardiovascular manifestation or the problem may be insidious and mild. Making the correct diagnosis requires considerable knowledge of the systemic nature of many of the connective tissue diseases.

This article reviews for the primary care physician the connective tissue diseases that produce or are associated with cardiovascular manifestations. Rheumatic fever and mucopolysaccharidoses are separate topics and are not discussed.

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Rheumatoid Arthritis

Pericarditis is the most common and best studied cardiovascular manifestation of **rheumatoid arthritis in adults**. Necropsy studies of patients with rheumatoid arthritis have revealed thickened pericardium with or without inflammation in up to 50% of patients, although very few had a clinical history of pericarditis. Most patients with cardiac involvement have high titers of rheumatoid factor, and about one-half have rheumatoid nodules. Echocardiography has demonstrated evidence of percardial effusion in about one-third of all patients with rheumatoid arthritis. Tamponade is rarely noted, but an abrupt decrease in systemic corticosteroids

may precipitate tamponade. Constrictive pericarditis occurs but is rare. Pericardial fluid is quite similar to pleural fluid, i.e., there is a very low glucose content, the LDH may be elevated, and cholesterol crystals may be found.

An interstitial or granulomatous myocarditis is noted in about 20% of adult patients with rheumatoid arthritis. The amount of inflammation does not correlate with the clinical status of the heart; granulomas may affect the conduction system, however. There is controversy over mitral valve involvement. A study at Massachusetts General Hospital over 20 years ago reported histologic evidence of valvular fibrosis in 25% of patients. However, there is a report of an increased incidence of mitral valve disease diagnosed by the echocardiogram as demonstrated by a reduced E to F slope of the anterior leaflet of the mitral valve, although later studies seem to disprove this. Incompetency of the aortic valve has been reported, secondary to granulomas in the valve itself, but this is unusual. Granulomas may also involve the base of the aorta.[1-5]

In adult patients with long-standing, seropositive rheumatoid arthritis, there may be a diffuse vasculitis, which is usually mild and associated with sensory-motor defects, but there can be a severe vasculitis which resembles polyarteritis nodosa. In this latter category, coronary vasculitis and deaths from myocardial infarction have been reported.

The only significant clinical cardiovascular manifestation of **juvenile rheumatoid arthritis** is pericarditis. It is seen mainly with the systemic form of the disease.[6]

Seronegative Spondyloarthritides

Cardiovascular involvement is a well-recognized manifestation of ankylosing spondylitis and Reiter's disease. Studies have shown about 3.5% of aortic involvement with these two diseases 15 years after the onset of musculoskeletal symptoms and after 30 years, the frequency rises to 10%. (There is confusion in the literature concerning the differentiation of patients with ankylosing spondylitis and Reiter's disease.) The main cardiovascular problems involve aortic insufficiency. The histology may be confused with that of luetic aortitis. One may see the aortic wall essentially destroyed. The intima may be thickened and fibrous. The elastic fibers may be fragmented, clumped and interrupted by fibrous tissue. The adventitia may be markedly thickened, fused with the media, and associated with thick-walled nutrient arteries. The conduction defects seem to arise from scar tissue extending from the aortic valve cusps to the interventricular septum, in the region of the Bundle of His. Congestive heart failure may be due to pulmonary hypertension secondary to anoxia from an inability to expand the chest.

Similar cardiovascular disease is noted in spondylitis of psoriasis and inflammatory bowel disease.[7-10]

Relapsing Polychondritis

Heart and aortic problems appear in up to 25% of patients with this rare disease. Most ominous is aortic insufficiency secondary to progressive dilatation of the aortic ring and ascending aorta. The histology is essentially identical to that of ankylosing spondylitis. In addition, there may be a "floppy" mitral valve. Aortic aneurysms may develop. On occasion there is coexistence of other connective tissue diseases, and there may be systemic vasculitis. HLA-B27 frequency is no more common than the general population. Attempts at replacing the aortic valve may be disastrous because of an inability of sutures to hold in the diseased tissue.[11]

Systemic Lupus Erythematosus

Clinical evidence of pericarditis occurs in 25% of patients with systemic lupus erythematosus. The incidence is not as common now as it was several decades ago because of the use of corticosteroids and other anti-inflammatory agents. Tamponade is unusual. The pericardial fluid is non-specific and is quite similar to viral pericarditis. In contrast to rheumatoid arthritis, therefore, the sugar content is not significantly low, but the pericardial fluid may contain LE cells and the anti-nuclear antibody titer on the fluid may be high. The incidence of pericardial disease at autopsy is somewhat hard to interpret since the data are somewhat clouded by studies done in the pre-corticosteroid era. A very high percentage of pericarditis is still noted at autopsy but some of the diseased pericardium could be secondary to uremia. Constrictive pericarditis rarely is noted.

Myocardial involvement is high—40% at autopsy. Electrocardiographic studies show that around 10% of patients have prolonged PR intervals. Coronary vasculitis occurs and myocardial infarctions secondary to this have been reported, but coronary artery disease with lupus could well be secondary to serum lipid abnormalities from the long-term use of corticosteroids.

All sorts of murmurs have been described. Aortic insufficiency occurs on rare occasions and is secondary to aortitis. In the pre-corticosteroid era, one-third of patients had Libman-Sacks endocardi-

tis, usually on the undersurface of the tricuspid and/or mitral valves, so that the position of the verrucae does not interfere with valve closure. Myocardial valve scarring, however, apparently can produce a diastolic murmur.

Drug-induced SLE may produce pericarditis and can produce tamponade. Most investigators consider drug-induced lupus as a separate entity rather than "subclinical lupus", which is unmasked by drugs.[12]

Progressive Systemic Sclerosis

Cardiovascular involvement is reported in about 15% of cases of progressive systemic sclerosis, or scleroderma. The most common type is pericarditis, which may be characterized by a silent effusion and mild dependent edema and is often misdiagnosed as the "edematous phase" of scleroderma.

Myocardial disease associated with systemic sclerosis may be of several types. Diffuse fibrosis is uncommon. Studies of sudden death in patients with systemic sclerosis have shown a proliferative intimal lesion in intramyocardial vessels and an unusual type of necrosis and fibrosis called "contraction band necrosis", probably secondary to intermittent ischemia with reperfusion. This sequence of events eventually leads to perivascular and band-like fibrosis, which some say can be differentiated from coronary artery disease.

It should be emphasized that the patient with systemic sclerosis who has angina and an abnormal electrocardographic tracing may have normal coronary angiograms, and so it must be remembered that scleroderma is a disease which causes severe loss of the microcirculatory bed. Because of this, coronary artery bypass grafts may not help since the lesion may be distal to the bypass and the myocardium remains under-perfused because of the extensive small vessel disease.[13,14] Pulmonary hypertension, with or without interstitial lung disease, may be a severe problem.

Polymyositis

Clinical cardiac involvement is said to be unusual in polymyositis, but it is probably more common than is generally thought. There may be arrhythmias, pericarditis, congestive heart failure, and death. Electrocardiographic abnormalities are noted in 30-50% of patients sometime during the course of the disease.

In childhood dermatomyositis, which is a different entity altogether and usually secondary to diffuse vasculitis, electrocardiographic abnormalities indicate a poor prognosis.

Histologic studies are quite similar to those of other skeletal muscles, i.e., inflammed muscle is noted along with necrosis and fibrosis.

Some of the published data on polymyositis may be clouded or inaccurate due to wrong diagnoses. Some of the cases previously reported as polymyositis may actually represent systemic lupus erythematosus or mixed connective tissue disease. The common cause of death in patients with polymyositis used to be hypoxia from chest wall and diaphragmatic muscle involvement.[15] Now, sepsis, malignancy and cardiovascular diseases are the main causes of death.

Mixed Connective Tissue Disease (Lupus, Systemic Sclerosis, and Polymyositis)

Pericarditis is the most frequent cardiac finding in mixed connective tissue disease and probably is the "lupus component." Cardiac problems in adult patients are less common than are pulmonary problems. In the childhood type, however, about two-thirds of patients reported had heart involvement, and in autopsies of three children who had no clinical heart problems there was myocardial necrosis, interstitial and perivascular inflammatory infiltration, interstitial fibrosis, and intimal thickening and narrowing.[16]

Werner's Syndrome

Werner's syndrome, or adult progeria, has been quite thoroughly studied. The afflicted patients usually are of short stature, have premature graying and baldness, scleroderma-like changes, cataracts, hypogonadism and diabetic tendency, along with calcification of the blood vessels, and rings and/or cusps of the mitral and aortic valves. The most common causes of death are vascular accidents and neoplasms. Osteoporosis may be a part of the disease. The T lymphocytes behave as those in a 90-year-old. One theory is that the T cell abnormalities probably are secondary to anti-lymphocytic antibodies.[17]

The Vasculitides

Cardiac involvement is clinically apparent in one-third of patients with **polyarteritis nodosa**, and postmortem studies have documented involvement in two-thirds. Most commonly noted is congestive heart failure from coronary vasculitis, hypertension, or both. Pericarditis is present in less than one-fifth of the patients and seems related to vasculitis. Conduction problems have been reported, and the nutrient arteries to the conductive systems are vulnerable to vasculitis. The sinoatrial system

seems more frequently involved than the atrioventricular system. The development of arrhythmias, including supraventricular conduction abnormalities, indicates a poor prognosis.[18,19]

In **hypersensitivity angiitis**, pericarditis is noted on occasion, but myocardial involvement is almost unheard of.

Cardiac involvement in **allergic angiitis and granulomatosis** clinically parallels polyarteritis nodosa. It is differentiated from it, however, by the frequency of pulmonary vessel involvement, vasculitis of various types and sizes, intra and extra-vascular granuloma formation, eosinophilic tissue infiltrates, and association with severe asthma and peripheral eosinophilia.[18]

Wegener's granulomatosis can involve any structure in the heart. Pericarditis, however, is the most common clinical manifestation. Around 30% of post-mortem examinations have shown granulomas or evidence of vasculitis in the heart. Rarely has there been a report of endocarditis, pancarditis or coronary arteritis. Arrhythmias from ischemia have been reported.[21]

Takayasu's arteritis, a very rare condition which mainly affects young women, is characterized by inflammation and stenosis of large and intermediate sized arteries with frequent involvement of the aortic arch and its branches. There are two main clinical stages: 1) an acute phase characterized by signs and symptoms of an inflammatory process, and 2) a chronic phase characterized by signs and symptoms of occlusion. Many patients in the latter phase walk with a face-down posture, which is thought to prevent transient decreases in visual acuity and narrowing of the visual fields.

Radial, ulnar and carotid arteries are the sites most frequently involved with Takayasu's arteritis. Absence of at least one arterial pulse is recorded for 98% of patients. Bruits are found in 86% of patients, with most being heard in the supraclavicular area. There may be a diastolic component indicating collateral flow. Raynaud's phenomenon occurs in only 10% of patients. About 50% have hypertension, which may be secondary to elevated renin levels. About one-half have syncopal episodes. Cerebral infarction occurs in less than 10% of patients. Both right- and left-sided congestive heart failure occurs and generally reflects systemic and pulmonary hypertension. Rarely is there aortic incompetence, myocarditis, pericarditis or coronary arteritis. Death occurs from the disease in about 10% of patients.[22]

Giant cell arteritis, the preferred name for what is sometimes called temporal or cranial arteritis, occurs primarily in patients over age 60 and is commonly associated with polymyalgia rheumatica. If there is evidence of cranial vasculitis, it is probably correct to infer that the aortic arch is involved. Radiographic and histopathologic studies are similar to those for Takayasu's disease. There may be angina or myocardial infarction. Rarely reported is aortic insufficiency, dissection, or rupture of the aorta. There may be hypertension from renal artery involvement.[23]

In **Kawasaki's disease**, also called juvenile polyarteritis and mucocutaneous lymph node syndrome, complete recovery is the rule, but a small number of patients may die four weeks after the onset of symptoms from a myocardial infarction due to coronary vasculitis. The greatest risk is for children less than one year of age. Electrocardiographic tracings are abnormal in 60% of the cases, usually show non-specific changes, and are of uncertain predictive value. However, coronary vasculitis seems to occur in greater than 50% of cases and may be associated with aneurysms. Fortunately, healing seems to occur in most of those affected.[24]

Thrombophlebitis is the most common cardiovascular manifestation of the rare **Behcet's disease**. There is an increased incidence of pericarditis, aneurysms and vascular occlusions.[25]

In **amyloidosis**, heart involvement occurs in all types of the disease, including amyloidosis associated with multiple myeloma. Signs and symptoms of right-sided congestive heart failure may dominate the early clinical picture. Chest radiographs may show only minimal cardiomegaly. Electrocardiographic tracings may show low voltage, conduction disturbances, arrhythmias, poor R wave progression, and patterns simulating old myocardial infarctions. Echocardiograms may show symmetrically thickened left ventricular wall, hypokinesia, and decreased systolic thickening of the inter-ventricular septum and left ventricular posterior wall, and a small or normal size left ventricular cavity. Amyloid may be deposited in the coronary arterioles and produce angina and myocardial infarction; suspect this diagnosis in an elderly patient with an idiopathic restrictive cardiomyopathy.[26] Rectal biopsy is diagnostic in most cases but transvenous endocardial biopsies have been employed safely to make an antemortem diagnosis. Percutaneous abdominal fat-pad needle biopsy shows promise as a safe and rapid method of diagnosis.

Heart involvement with **sarcoidosis** is usually asymptomatic but postmortem studies have shown involvement in up to 20% of patients. Around 10% are said to have a conduction or rhythm problem.[27]

Disorders of Collagen Structure and Metabolism

There are several different types of **Ehler's-Danlos syndrome**, and the genetic defects have been worked out in some of them. Cardiovascular manifestations include multiple aneurysms, aortic rupture or major arterial tears, arteriovenous malformations, varicose veins, cardiac septal defects, aortic insufficiency, and mitral and/or tricuspid valve prolapse. Microscopic studies of arteries reveal surprisingly, only such subtle changes as separation of the elastic laminae or separation of the smooth muscle cells.[28]

Up to 60% of persons believed to be affected by **Marfan's syndrome** have auscultatory evidence of either mitral or aortic insufficiency or systolic clicks. In the classic case, average life expectancy is halved. Echocardiographic studies have shown that 80% of patients, irrespective of age or sex, have prolapse of at least the posterior mitral leaflet; the prolapse is often pansystolic, with exaggerated leaflet excursion suggesting valvular redundancy. Various arrhythmias have been noted. Death from aortic disease seems to be the main cardiovascular problem. There may be aortic insufficiency from a greatly widened ring, myxomatous degeneration of the valve itself, or dissection associated with cystic medial necrosis. In addition, there may be sinus of Valsalva aneurysms. Microscopic examination of the aorta reveals scar tissue in the media, cystic medial degeneration, and a marked disruption, fragmentation, and sparsity of elastic fibers.[30]

Homocystinuria results from various defects in the metabolism of methionine but most commonly from a deficiency of cystathionine synthetase. Skeletal abnormalities and lens dislocation lead to confusion with Marfan's syndrome. With homocystinuria, however, there is neither arachnoidactyly nor hypermobile joints. The cardiovascular abnormalities are limited to blood vessels. There is frequently a severe thrombotic diathesis. This results from disruption of the vascular endothelium, exposure of underlying collagen, and subsequent platelet aggregation.[31]

According to the literature, **osteogenesis imperfecta** is about as common as Marfan's syndrome and its cardiovascular manifestations are similar but occur less frequently.[31,32]

An increased incidence of arteriosclerosis and hypertension has been reported in patients with **pseudoxanthoma elasticum**. There is also a very high incidence of mitral valve prolapse.[33]

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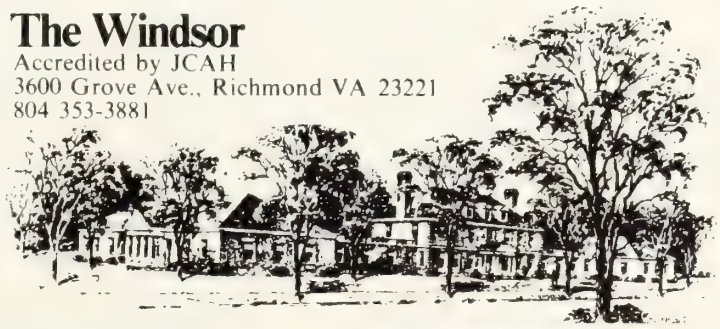

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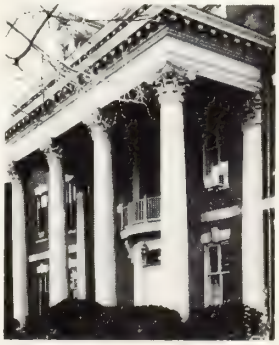

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Coronary Spasm Affecting Result of Bypass Operation: Case Report

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During the evolution of concepts related to angina pectoris, varying emphasis has been given to either coronary spasm, thrombosis, or arteriosclerosis as the source of obstruction. William Heberden described spasmodic pains of angina;[1] Peter Latham related these spasms to coronary arteries;[2] and Sir William Osler proposed a mechanism by which coronary artery spasm produced angina through disturbed cardiac function and stretching of myocardial tissue.[3] Despite these impressive beginnings, the consistent findings of fixed, sclerotic stenoses at postmortem examination after angina pectoris[4] led to the disregard of spasm as a mechanism of ischemic cardiac pain until recently.

Interest was revived in coronary artery spasm in 1959 as the result of Prinzmetal's description of variant angina.[5] During the 23 years following this report, vasomotion of the coronary arteries has been increasingly implicated as an important component of ischemic chest pain syndromes,[6] including such diverse clinical presentations as unstable angina,[7,8] "typical" exertional angina,[9] and myocardial infarction.[10,11] Each of these syndromes is usually associated with demonstrable sclerotic arterial obstructions,[12] but their occasional ocurrence with slight or no coronary sclerosis[13-15] suggests in addition a varying role of coronary spasm.

We present a person in whom the combination of severe obstructive coronary artery sclerosis in a single vessel and spasm were both clinically significant.

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Case Report

A 46-year old woman was seen in June 1978 with complaints of both exertional and resting angina refractory to beta blockade and nitrate therapy. She had a prolonged episode lasting 2-3 hours in December 1977, with anterolateral T-wave inversion in her electrocardiogram (leads I, aVL, V1-V4). Risk factors for coronary artery disease included cigarette smoking, a positive family history, and mild hypertension. Cardiac catheterization demonstrated anterior wall hypokinesis and a 90% fixed, proximal obstruction in the left anterior descending coronary artery (Fig. 1A).

Despite intensive drug treatment, chest pain persisted, and reversed saphenous vein bypass grafting to the left anterior descending (LAD) coronary artery was performed. Although the procedure was technically successful, the postoperative hospital course was characterized by recurrent hypotension, ventricular arrhythmias and chest pain. These were associated with inferior wall ST segment elevation in the ECG, ultimately controlled by infusion of intravenous nitroglycerin.

In spite of concern over possible graft closure, the patient became stable and was without symptoms for six months, when exertional and rest angina returned. Beta blockade and nitrates proved ineffective, and she returned to the hospital in February 1979. Exercise test to 7 METS and 74% age-predicted maximum heart rate was negative, including thallium myocardial imaging. Cardiac catheterization showed no change in the native coronary vessels (single 90% proximal LAD lesion), and a patent bypass graft. It was concluded that the pain was most likely non-cardiac in origin, although the possibility of coronary artery spasm was considered.

Her chest discomfort then remained quiescent for almost two years, when progressively severe symptoms prompted another admission in February 1981. Episodes usually took place in the morning at

rest, although exertional pain also occurred. Thallium exercise to 9 METS and 74% age-predicted maximum heart rate was again negative, save for equivocal ECG changes. Cardiac catheterization was unchanged from the study in February 1979, disclosing only a single LAD stenosis and a patent bypass graft (Fig. 1B). The right coronary artery was normal (Fig. 2A).

Ergonovine maleate provocation testing was performed. Following the administration of 0.2 mg intravenous ergonovine the right coronary artery (RCA) became totally occluded proximally (Fig. 2B). This was accompanied by marked ST segment elevation in the inferior surface ECG leads, and the patient's typical chest pain. Intracoronary nitroglycerin totally relieved the chest pain, reversed the ECG changes, and restored normal flow to the RCA (Fig. 2C).

The demonstration of coronary artery spasm resulted in discontinuation of nitrates and beta blockers and institution of therapy with nifedipine. Subsequently there has been a marked reduction of angina (except during periods of attempted drug withdrawal), normalization of exercise tolerance, and an absence of adverse reactions to the medication.

Comment

Both coronary artery spasm and a sclerotic coronary obstruction contributed to this patient's chest pain and signs of ischemia initially in the LAD region. The 90% proximal LAD stenosis produced significant hemodynamic effects, as evidenced by anterior ECG changes and anterior wall hypokinesis prior to coronary bypass. Operation relieved the sclerotic component of the myocardial ischemia and inadvertently displayed the spastic component in

Fig. 1A. Left anterior oblique view of left coronary system. Constriction estimated at 80%-90%.

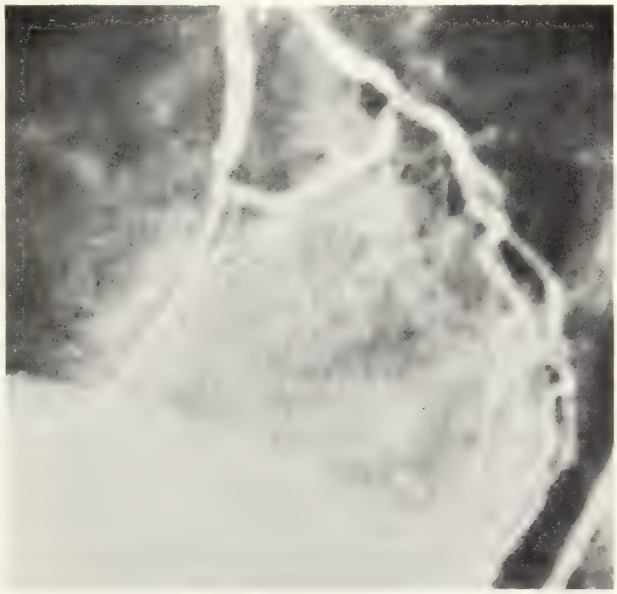

Fig. 1B. Two years later, system unchanged except for added patent saphenous vein graft.

Fig. 2A. Right coronary artery before ergonovine. No major obstructions.

Fig. 2B. During ergonovine, spastic obliteration of artery coinciding with pain.

Fig. 2C. Normal flow restored to artery by intracoronary nitroglycerin.

the absence of palliative medications. Ergonovine maleate later confirmed the clinical impression of coronary spasm. At this time the marked ST elevation in the inferior surface ECG leads corresponded to the transient obliteration of the right coronary artery seen on angiogram.

Separation of the sclerotic and spastic features demonstrates the importance of each in the production of angina, and illustrates the concept that both mechanisms may contribute to ischemia in an unknown percentage of patients with symptomatic coronary artery disease. Bertrand and colleagues used ergonovine in 1,089 patients undergoing coronary arteriography;[16] they elicited spasm more often in persons with angina at rest, after recent infarction, and with coexistent arteriosclerosis.

A role of coronary spasm in persons with acute coronary insufficiency syndromes without major obstructive lesions has often been accepted on the basis of responses to nitrates or, more recently, calcium antagonists, such as nifedipine. Ergonovine maleate provocation in the appropriate clinical setting has been described as a highly sensitive and specific means to diagnose coronary artery spasm.[17] Additionally, ergonovine testing predicts the initial response to treatment with calcium channel blockers[18] and detects spontaneous clinical remissions and the need for continued therapy.[19] Relatively small numbers of patients have been tested with ergonovine, however, because of both the requirement of catheterization and uncertain safety. Safety considerations may possibly restrict its use to persons with either normal coronary arteries or a single stenotic lesion.[20] There are documented risks associated with provocation testing, including cardiac arrhythmias, myocardial infarction and death.[21]

The limitation of ergonovine provocation as a test for coronary spasm may lead simply to more widespread trials of calcium antagonists in persons suspected of a significant component of coronary spasm. In the absence of knowledge about coronary anatomy, that could be an appropriate response for persons with angina in a setting of a favorable exercise test and relative freedom from other indications of arteriosclerosis. On the other hand, angina at rest may imply either coronary spasm, or extremely severe sclerotic obstructions, or other paroxysmal events causing increased myocardial oxygen demand, such as dysrhythmias. The latter possibilities may require early study rather than empirical trial of a calcium channel antagonist. Although a significant role of spasm and effectiveness of vasodilator may be inferred when fixed obstructions are found to be few or mild, benefit from calcium antagonists has also been reported despite multivessel obstructive disease.[22]

In persons whose clinical disability seems out of proportion to the severity of anatomic coronary sclerosis, the possibility of coronary artery spasm should be considered. Either ergonovine maleate provocation testing or a therapeutic trial of a calcium channel blocking agent would be appropriate. Our case report underscores the dynamic interrelationship of atheromatous coronary stenosis and vasospasm and illustrates the beneficial effects of calcium channel blocking agents in selected patients.

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Morbidity and Mortality Conference: Coronary Artery Disease and Surgical Bypass

From the Medical College of Virginia/Virginia Commonwealth University.
Case Presentation by John L. Leahy, MD.
Clinical Discussion by George W. Vetrovec, MD.
Pathology Discussion by Fergus O. Shiel, MD.

Presentation of Case

Dr. John L. Leahy: A 65-year-old retired white male construction worker was transferred to the Medical College of Virginia Hospitals for coronary bypass surgery.

He was well until three months prior to admission when he suffered an attack of painful "shingles" over the lower abdomen. Subsequently he noticed dull left arm pain radiating into his chest whenever he was dancing or playing golf; these were relieved by rest. One week prior to admission the attacks of pain increased in frequency and occurred with less exertion. Two days prior to admission he awoke at 5:30 am with left arm pain radiating into his chest; this was relieved in one-half hour by an analgesic. The following day he again awoke at 5:30 am with the same left arm and chest pain not relieved by an analgesic and for which he was hospitalized.

On admission to a local hospital he was described as being in no acute distress and appearing younger than his stated age. His temperature was 98°F, blood pressure 160/92, pulse 64/min and regular. On cardiac examination no murmur or gallop was noted. The abdominal aorta was palpable and all peripheral pulses were present.

His electrocardiogram showed deep inverted T waves in precordial leads V2 through V6 (Fig. 1). A suspected acute myocardial infarction was excluded by normal cardiac enzymes with a peak SGOT of 22, LDH of 132, and CPK of 96 U/l (all normal values). His EKG did not appreciably change. Despite increases in propranolol, isosorbide dinitrate and nitroglycerin ointment, the patient continued to experience chest pain while at rest; the pain was relieved by nitroglycerin. On the fourth day he underwent coronary angiography, at which time 85% proximal left anterior descending and circumflex lesions were demonstrated with good distal runoff (Fig. 2). The right coronary artery showed a 50% mid-proximal eccentric lesion (Fig. 3). A left

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Submitted October 1982.

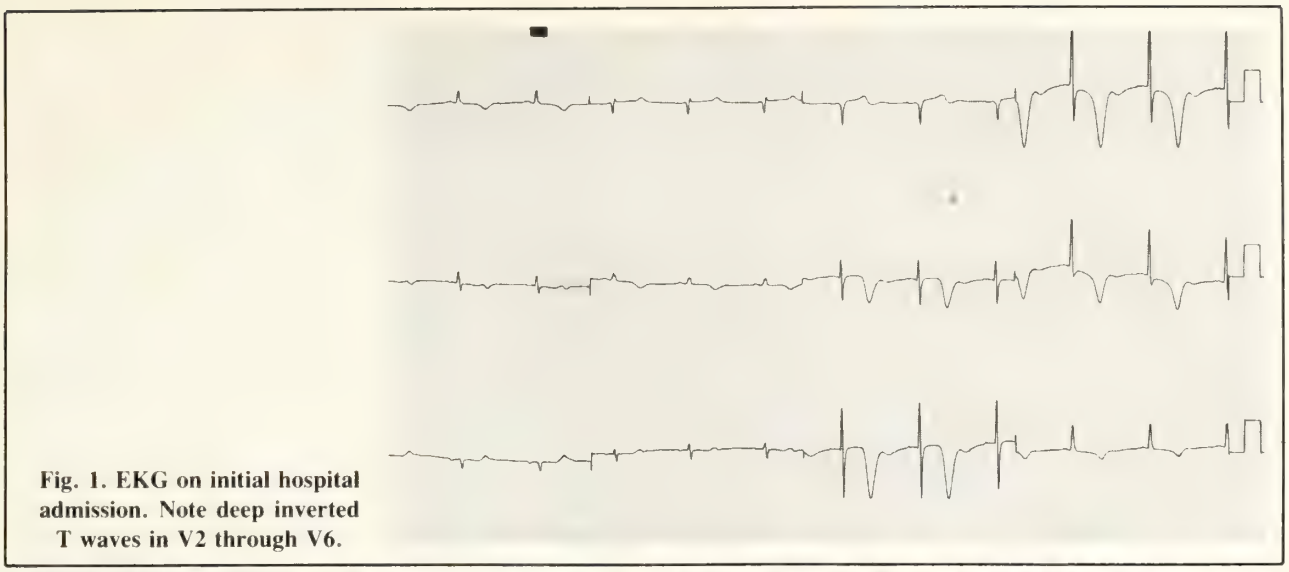


Fig. 1. EKG on initial hospital admission. Note deep inverted T waves in V2 through V6.

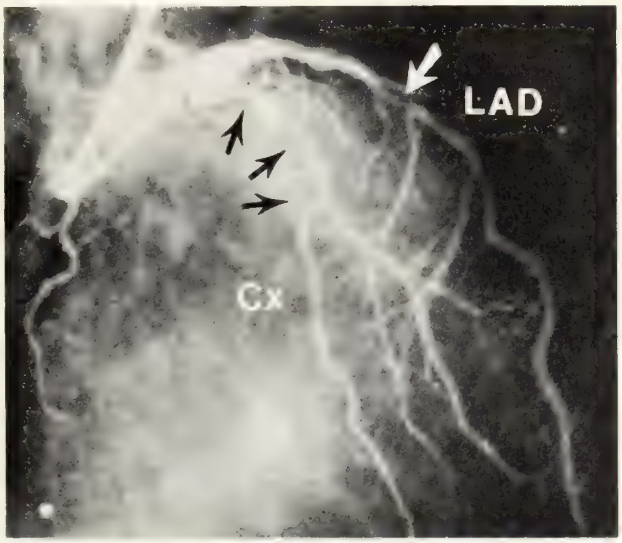


Fig. 2. Coronary angiography. White arrow = critical 85% left proximal anterior descending lesion. Dark arrows = three serial circumflex lesions.

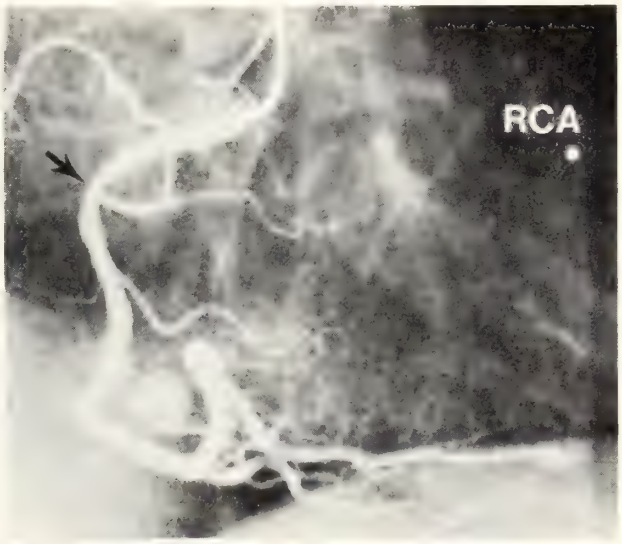


Fig. 3. Selective right coronary injection. Arrow = 50% proximal mid-juncture lesion.

ventriculogram showed anterior hypokinesis with overall normal ejection fraction.

He experienced further episodes of chest pain at rest relieved by nitroglycerin, and on the seventh hospital day he was transferred to MCV for coronary bypass surgery. His medications at the time of transfer were propranolol 60 mg orally every 6 hours, isosorbide dinitrate 40 mg orally every 6 hours, nitrol paste 2 inches every 6 hours as needed.

His past medical history included a diagnosis of hypertension (noted eight months prior to transfer), treated with diuretics and low-dose beta blockers. There was no prior history of diabetes mellitus, hyperlipidemia or obesity. There was no family history of heart disease.

On admission to MCV, physical examination revealed a thin, well-developed male with a pulse of 60/min and regular, blood pressure 150/78 and respirations 20/min. His neck veins were flat. A left carotid bruit was heard. His lungs were clear to auscultation. No murmurs were heard, but an atrial gallop was noted. A 4-to-5-cm pulsation was palpable in the epigastrium, with an associated bruit. Bilateral femoral pulses with bruits were noted. On the left there were no pulses below the femoral. No edema was present.

The EKG revealed sinus bradycardia and T-wave inversions of the anterior lateral leads consistent with ischemia (Fig. 1).

He continued to have daily chest pain relieved by sublingual nitroglycerine until he underwent bypass

surgery on the third MCV hospital day. At surgery, four aortocoronary saphenous vein grafts were inserted to distal sites including the right coronary artery, the left anterior descending coronary artery and two marginal branches of the circumflex artery. Immediately following completion of the grafts, severe hypotension and ventricular arrhythmias developed which could not be controlled and the patient died in the operating room.

Radiography

Dr. Karsten F. Konerding: This patient's preoperative chest radiograph showed a normal-size cardiac silhouette and moderate tortuosity of the descending aorta. The lungs are normal. Supine and lateral abdominal radiographs show no evidence of a calcified aortic aneurysm, or of any other masses.

Discussion

Dr. George W. Vetrovec: In order to discuss the decision-making process regarding medical or surgical management of coronary artery disease, an understanding of coronary anatomy as defined by coronary angiography is necessary. Blood supply to the heart is via the right and left coronary arteries. The left main coronary artery supplies a major portion of the left ventricle by two major vessels: the left anterior descending (supplying the anterior wall) and the circumflex (supplying the lateral wall). The right coronary artery distributes blood to the inferior left ventricular wall. The terms one-, two- or three-vessel disease refer to significant disease involving any one of these vessels, the left anterior descending, circumflex and/or right coronary arteries, while the term "left main disease" refers specifically to the short left main trunk prior to its bifurcation into the anterior descending and circumflex vessels.

The anatomic severity of coronary disease is useful in defining prognosis and potential for coronary bypass surgery. It is important to recognize that clinical symptoms of angina correlate poorly with the anatomic severity of obstructive coronary artery disease.[1] Patients with severe diffuse coronary artery disease may have little or no symptoms, while patients with more limited but significant disease have severe symptoms.

Early in the history of coronary angiography, it became apparent that coronary angiography provided excellent prognostic data for patients with coronary artery disease. Longitudinal data from the Cleveland Clinic illustrates this fact.[2] Of 490 patients who underwent coronary angiography without subsequent bypass surgery, the cumulative survival over a 10-year followup period was directly related to the severity of anatomic disease. Patients with single-vessel disease had a cumulative 10-year mortality of 35%, compared to a 65% cumulative mortality for patients with significant three-vessel or left main disease. The same study of patients with coronary artery disease demonstrated a correlation between prognosis and left ventricular function. Those patients with normal left ventricular function had a 10-year cumulative mortality of 35%, compared to a 75% mortality for those with diffuse left ventricular dysfunction. Patients with limited scars or discrete aneurysms had an intermediate cumulative mortality. Thus, the more extensive the anatomic coronary artery disease or the worse the left ventricular function, the poorer the prognosis.

Coronary angiography may also establish the potential for coronary bypass surgery. Anatomic features consistent with favorable surgical anatomy include tight proximal stenoses; large, relatively disease-free, distal vessels and normal left ventricular function. Patency of bypass grafts appears to be related to blood flow within distal vessels; if the distal segments are severely diseased, a much higher incidence of graft occlusion occurs than if there is good distal runoff. Likewise, poor left ventricular function increases the risk of a bypass operation.

Medical vs Surgical Management

Relative to symptomatic benefit, there is little doubt that bypass surgery affords excellent clinical improvement for most patients possessing favorable surgical anatomy. Repeated studies indicate that regardless of the parameter of functional status chosen for assessment, patients undergoing bypass have less angina, greater exercise tolerance and less medication requirements than their medically treated counterparts. Mathur and Guinn's[3] study of 100 patents, randomized to medical or surgical treatment and followed for 24 months, illustrates this point. Their results show a significant improvement in exercise test time for the surgically treated group beginning nine months post surgery and persisting for 24 months. This and many other studies document the repeated symptomatic benefits of surgery for patients with angina pectoris.

A more difficult question to answer concerns longevity benefits from bypass surgery. I believe there are important anatomic factors that predict which patients are likely to have improved survival following bypass surgery. Patients with the greatest

amount of viable myocardium at jeopardy because of coronary artery disease are those most likely to experience increased survival following surgery.

The subgroup of patients with left main disease is defined best. Takaro et al[4] in the Veterans Administration Cooperative Study showed that patients with critical left main disease randomized to bypass surgery had a significantly lower ($p < 0.01$) three-year mortality (7%) as compared to that (20%) of their medically treated counterparts. Furthermore, those patients who had critical right disease as well as left main disease were most benefited. (The latter fact supports the concept that the more myocardium in jeopardy, the greater the potential benefit from bypass surgery.) Significant left main stenosis comprises 5%-10% of all patients with angina undergoing catheterization, and there is general agreement that such patients are best treated surgically.

After left main disease, longevity benefits of surgery become more difficult to predict. The VA Cooperative Study of chronic stable angina suggests that for at least 36 months no significant difference exists between medical and surgical treatment for patients without left main coronary artery disease. However, in that study a subgroup of surgical patients with significant three-vessel disease and segmental left ventricular wall motion abnormalities enjoys an improved survival.[5] Kloster[6] reports no significant difference for "coronary events" (cardiac death, myocardial infarction or recurrent unstable angina) between medical and surgical treatment for 100 randomized patients. However, the subset of those patients with significant three-vessel disease undergoing bypass surgery had a significantly lower incidence of coronary events compared to those treated medically. Recent five-year followup data from the European Cooperative Study,[7] a randomized multicenter trial of medical-versus-surgical management of chronic stable angina, reveal a significant improvement in survival for all patients undergoing coronary bypass operations. However, careful examination of this data demonstrates that significant longevity benefits from surgery occurred only in those patients with left main disease or three-vessel disease.

Thus, patients with extensive coronary artery disease, rendering the largest amount of functional myocardium at jeopardy, are the ones most likely to experience low mortality and morbidity as a result of bypass surgery. Although anatomic features are important in surgical decisions, other clinical factors are also, e.g., the patient's symptomatic response to medication; side effects, if any, to medications; age and overall long-term exercise goals.

Uses of Coronary Angiography

When does one consider doing coronary angiography? Patients unresponsive to standard medical management or who have intolerable side effects are considered for angiography in the hopes of demonstrating potentially bypassable disease. In this group, the symptomatic benefits from surgery are clear.

Next, patients with unstable angina are considered. These patients frequently have three vessel coronary artery disease, and approximately 10% have significant left main disease. Furthermore, these patients often have recurrent bouts of unstable angina despite medical treatment[8] and an incidence of 10%-18% normal coronaries, which is important diagnostically and prognostically.

Patients with atypical or Prinzmetal type angina also should be considered for angiography. Approximately half of patients presenting with transient ST segment elevation and pain at rest without infarction have significant focal coronary obstructions. The other half have coronary artery spasm with otherwise normal coronary arteries.

For more on coronary artery spasm, see page 432.

Other indications for considering catheterization include patients with syndromes of chest pain of unknown etiology in which standard noninvasive studies have not established a diagnosis. In certain individuals non-invasive studies may suggest severe coronary artery disease. For example, 2 mm of flat ST segment depression during non-maximal treadmill exercise testing, particularly when associated with a drop in exercising blood pressure, suggests severe coronary artery disease. In addition, ST segment elevation during exercise may predict relatively severe anatomic coronary artery disease.

Patients with persistent congestive heart failure following myocardial infarction also are considered for catheterization. Here one looks for surgically treatable mechanical abnormalities, including left ventricular aneurysm, acute mitral regurgitation and rarely ventricular septal defects. The latter two are associated clinically with systolic murmurs. Patients who have diffuse left ventricular dysfunction without a localized scar generally are not good candidates for bypass operations, as coronary revascularization provides little benefit for resting left ventricular function and the surgery itself carries an increased risk.

Patients who have recurrent angina post-coronary bypass surgery may be considered for repeat angiographic studies. Those with early recurrent

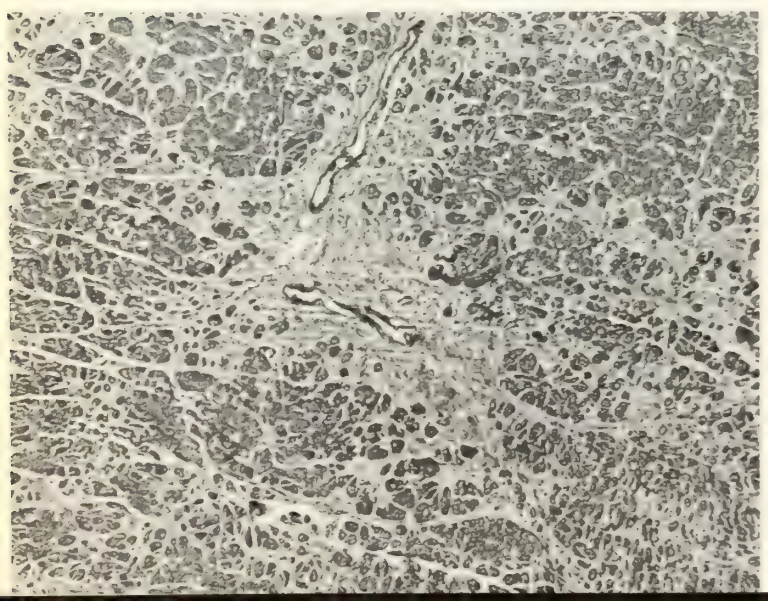
symptoms generally have graft closure. Those with late recurrent angina frequently have evidence of progressive native coronary artery disease, either in vessels beyond graft insertion or in previously ungrafted vessels. In early recurrent angina, if the reconstructive anatomy was favorable, catheterization may be useful in excluding a surgical error which may be correctable. If the reconstructive anatomy was generally unfavorable, repeat angiography or surgery may not be considered, as the chance for long term graft patency appears small. In late recurrences of angina, angiography may select patients who are candidates for a second bypass because of progression of coronary artery disease.

Followup

Followup of sucessful vein grafts is of considerable interest. As noted above, early occlusion of vein grafts is usually related to poor distal runoff or technical error. However, patients with recurrent symptoms beginning more than 2 to 3 months after surgery usually have evidence of progression of their native coronary artery disease. Campeau, in an extensive study of patients undergoing bypass grafting with repeat early and late angiograms suggests that late graft closure is much less common than is progression of disease in previously ungrafted vessels and occurs less often in the vessel beyond graft insertion.[10] This is an important factor when advising patients post-operatively.

Fig. 4. Whole mount of left anterior descending artery 90% occluded by atherosclerotic lesion. (H&E stain)

Fig. 5. Histologic section of myocardium. Note multiple areas of ischemic fibrosis (H&E stain, × 100)



Regarding the patient presented today who had a 50% right coronary artery lesion, I believe this vessel should have been grafted as well as the more severely diseased left coronary artery, for the following two reasons. First, since recurrent symptoms may be related to progression of disease in non-grafted vessels, grafting may have prevented future angina. Certainly, prophylactic bypassing of undiseased vessels is not recommended, but it would seem prudent to bypass vessels with early and near critical lesions because of the progressive nature of the disease. Second, pathological studies suggest that in many instances angiography underestimates the degree of obstruction. This has been shown by Roberts in correlating angiographically demonstrated stenosis with pathological findings.[11] Stenoses, particularly in the range of 50%, are likely to be underestimated. Thus, it was appropriate to consider bypassing the right coronary artery lesion to assure adequate revascularization, especially since the patient was a good candidate for bypass surgery on the basis of his coronary artery anatomy, which revealed proximal stenoses, good distal runoff and reasonable left ventricular function. In addition, medical management failed to control symptoms. His death was unexpected and unfortunate, as the overall risk of bypass surgery in most institutions performing a large number of procedures is 1%-3%.

Pathology

Dr. Fergus Shiel: Autopsy permission was restricted to examination of the heart and great vessels. The heart weighed 580 gms (normal 250-350) and left ventricular hypertrophy (mural thickness, 15 mm) was present. No gross evidence of old or recent infarction was seen; however, atherosclerotic lesions were present in all three major coronary vessels. The most severe narrowing was seen in the left anterior descending (Fig. 4) and circumflex branches with lesser changes in the right coronary. Histological cross-sections of these vessels were studied on a Zeiss video plan image analysis sys-

tem. By this method the residual lumen area of the left anterior descending and circumflex branches measured only 10% of the original caliber. The right coronary artery was 50% narrowed at the point of maximum stenosis. Coronary artery bypass vein grafts to the left anterior descending, circumflex branches and right coronary arteries were all intact and patent.

Histological sections of the myocardium revealed hypertrophy of myocytes with multiple microfoci of ischemic and interstitial fibrosis (Fig. 5). There was no histological evidence of recent infarction. The immediate cause of death was likely an arrythmia.

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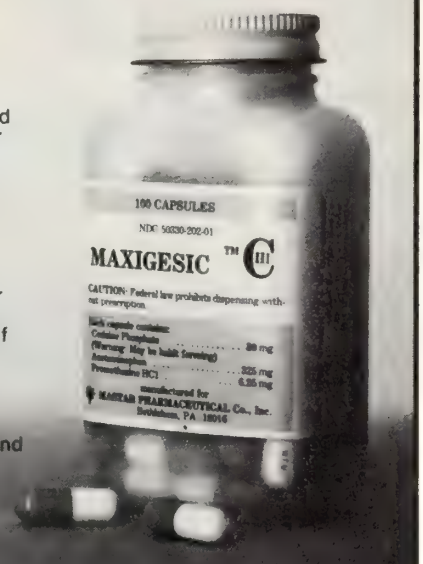

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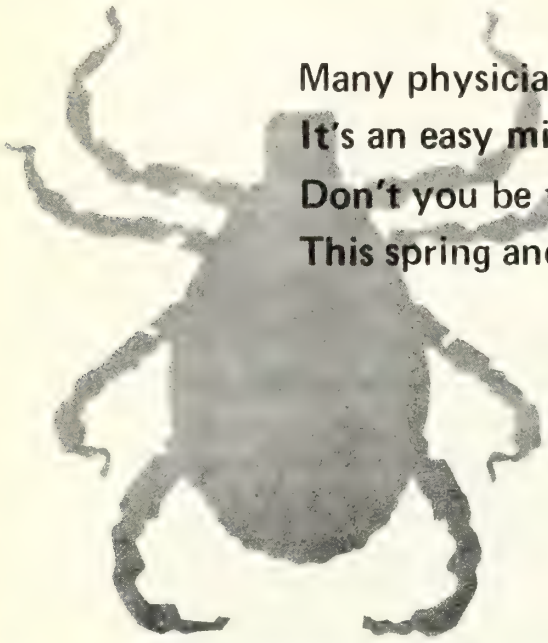

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THINK RMSF

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FEVER
MYALGIA

When the rash appears, it's getting late-

This message a service of the Virginia Chapter, National Foundation for Infectious Diseases, and The Medical Society of Virgi

Rocky Mountain Spotted Fever: a Tough Case to Diagnose

Case Report by Charles C. Ashby, MD,
Dinwiddie, Virginia
Discussion by Carl W. Armstrong, MD,
Richmond, Virginia

Case Report

On May 8, 1982, a 21 year old white woman was admitted to the hospital with a five day history of treatment with amoxicillin for an illness characterized by fever, pharyngitis and purulent nasal discharge. Two days after beginning treatment she noticed a generalized rash. When seen on the day of admission she was complaining of a headache and gave a history of having removed a tick approximately one week earlier.

On examination she was febrile. A faint maculopapular rash was present primarily on the trunk; the palms and soles were not involved. Some nuchal rigidity was noted. The following tests were normal: chest X-ray, complete blood count, heterophil, coagulation screen, blood cultures, cerebrospinal fluid (CSF) cell count and culture. A Proteus OX-19 titer was 1:40 and, when repeated, 1:80.

Tetracycline 500 mg, by mouth, four times a day was begun. Within two days she was afebrile and the rash cleared. On the sixth hospital day she was sent home to complete a ten-day course of tetracycline. Convalescent serum, obtained 25 days after onset, was paired with acute serum drawn three days after admission and sent to the state laboratory for Rocky Mountain spotted fever (RMSF) indirect fluorescent antibody (IFA) and complement fixation (CF) titers. Both tests demonstrated a four-fold rise in titer: the IFA titer rose from 1:2048 to 1:16,384 and the CF titer from 1:64 to 1:512.

Discussion

This case report illustrates several important points regarding the clinical diagnosis of Rocky Mountain spotted fever. First, the illness may mimic other disease processes and lead to a delay in appropriate therapy unless the physician has a high index of suspicion for RMSF. In this case, the clinical presentation could have been attributed solely to an upper respiratory infection complicated by sinusitis and amoxicillin allergy with rash. Indeed, the rash was more typical of allergy than of Rocky Mountain spotted fever; the rash of RMSF is initially macular, spreads centripetally, and may become petechial. In this case, the early peripheral lesions of RMSF may have been missed or absent. Occasionally no rash is noted in patients with RMSF.[1] The clues to the diagnosis were the history of a tick bite, the time of year, and the knowledge that RMSF is endemic in Virginia.

The second point illustrated is the occasional need to rule out meningitis when Rocky Mountain spotted fever is suspected. Nuchal rigidity is sometimes noted in patients with RMSF, and lumbar puncture frequently demonstrates the presence of 1-20 red blood cells per mm^3, presumably due to invasion of CNS blood vessels by *R rickettsii*.[2] The presence of fever, headache, nuchal rigidity and a petechial rash require that the CSF be examined and that cultures of CSF and blood be performed to exclude the possibility of bacterial meningitis, especially that due to *Neisseria meningitidis*.

The final point illustrated by this case is that treatment for Rocky Mountain spotted fever must be initiated based on clinical suspicion alone. Serologic tests depend on the demonstration of a four-fold rise in titer and are therefore only of use in confirming the diagnosis retrospectively. In the case presented, note that the acute OX-19 titers were not even suggestive of RMSF. In addition, the Weil-Felix test lacks specificity and has been replaced in reference laboratories by the IFA and CF tests.[3] The one test which shows promise for the early diagnosis of Rocky Mountain spotted fever is the skin biopsy with fluorescent antibody staining for rickettsiae, but this test is currently available only in some research laboratories.[4]

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Efficacy of Lengthy Therapy in Childhood Acute Lymphoblastic Leukemia

N. L. Dunn, MD, E. C. Russell, MD, N. B. McWilliams, MD, and H. M. Maurer, MD, *Richmond, Virginia*

A review of the literature reveals that children with ALL treated for three years have a relapse rate similar to those treated for two or three years more. Based on these figures and increasing reports of the potential toxicities of anti-leukemic chemotherapy, the authors conclude that there appears to be no benefit in the lengthier treatment period.

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Submitted 6-1-82.

MODERN treatment for childhood acute lymphoblastic leukemia (ALL) can now be expected to result in a 50%, five-year survival rate.[1] Recent refinements include treatment modifications based on the subclassification of ALL by clinical and immunologic criteria[2-4] in an effort to improve survival primarily by intensifying treatment in those patients initially identified as being at increased risk of early relapse. An important and controversial question currently being asked is how long therapy should be continued in order to provide maximum anti-leukemic benefits while minimizing potential acute and chronic toxicities from the treatment itself.

In an attempt to answer this question, we reviewed data on our ALL patients for the years 1965-1975, with special reference to those surviving more than three years.

Patient Population

The charts of 82 children in whom acute lymphoblastic leukemia was diagnosed between 1965-1975 were reviewed. The cut-off of 1975 was used to ensure a minimum followup of five years on all patients. There were 47 males and 35 females (M:F = 1.34:1) diagnosed between the ages of one day (the only case of congenital leukemia) and 17 years. Fifteen (18%) of the patients were black. Fifty-nine percent were considered at increased risk of early relapse based on criteria of the Cancer and Acute Leukemia Group B (CALGB): initial age (≤ 2 or ≥ 8 years) or white count ($\geq 30{,}000/mm^3$). Cell surface marker evaluation was not carried out on patients diagnosed during these years.

Therapy was not uniform during the ten-year time span studied, although 94% of the patients were treated on one of four CALGB protocols (6601, 6801, 7111, or 7411). Sixty-two (74%) patients received central nervous system prophylaxis with either intrathecal methotrexate alone or in combination with CNS irradiation. Two patients developed CNS relapse before prophylaxis could be instituted although no patient had overt CNS disease at the time of diagnosis.

Results

Seventy-eight (95%) of the children achieved remission status. Forty (49%) patients remained in complete continuous remission at 3 years. This group included 18 males (45%), two (5%) blacks, and 21 (52.5%) increased-risk patients. With the exception of two patients (treated for 2.5 and 11 months) all others continued on therapy between the third and fifth years post-diagnosis. During this

time period, three males and three females relapsed (15% of those in complete continuous remission at three years). The sites of initial relapse were the central nervous system in five (four had received CNS prophylaxis) and a simultaneous marrow and testicular relapse in the sixth. Five were initially increased-risk patients but otherwise had no particular identifying features. One child died of varicella complications between the third and fifth years of treatment, in autopsy-proven CCR.

Thirty-three (40%) children remained in complete continuous remission at least five years from diagnosis. This group included 15 (45%) males, two (6%) blacks, and 16 (48%) increased-risk patients. Treatment was continued for a median of 61 months (range 2.5-75 months). Followup times off therapy range from six to 178 months (median 36+ months). Total survival durations range between 61-180 months (median 99+ months). One male, treated for 60 months, relapsed in the marrow 16 months after therapy ended.

The overall relapse rate of those surviving complete continuous remission for at least three years is thus 17.5% (7/40) with a minimum of 24 months of additional followup available on all patients.

Table 1. Comparison of childhood ALL Relapse Rates in Three Series.

Series	All relapses		Testicular relapses	
	#	%	# males	% males
CCSG (randomized) overall	15/101	14.9	1/59	1.7
off at 3 years	15/57	26.3	1/37	2.7
on til 6 years	3/44	6.8	0/22	0
SWOG (randomized) overall	20/83	24.1	9/40	22.5
off at 3 years	11 40	27.5	7/17	41.1
on til 6 years	9/43	20.9	2/23	8.7
CURRENT SERIES overall	7/40	17.5	1/18	5.6
on til 5 years	6/40	15	1/18	5.6
off at 5 years	1/33	3	0.15	0

Discussion

As survival statistics for childhood ALL continue to improve, two key issues/goals have emerged: 1) further improvement in those statistics, primarily through the early identification and intensification of therapy for the increased-risk patients, and 2) minimizing potential acute and chronic toxicities associated with treatment,[5-7] particularly in those patients destined to be long-term survivors. The optimal time(s) to treat patients in order to achieve these goals has yet to be determined.

Increasing reports of series of patients coming off anti-leukemic therapy are now appearing in the literature.[8-14] Interstudy comparisons are difficult because of differing patient populations, lengths of therapy and, primarily, because of wide variations in treatment protocols, even within single series. Certain trends, however, have emerged. Most patients reported have been treated for two to five years (the majority for three years) and relapse rates off therapy have varied between 13.8-27.5% (mean 18.75%).[10-14] Most series have found that the majority of relapses occur in the first year off therapy and that males relapse more frequently than females even when testicular relapses are excluded.

The Children's Cancer Study Group (CCSG)[12] and the Southwest Oncology Group (SWOG)[11] are conducting a randomized study of three versus six years of maintenance therapy. Different treatment protocols are used by the two groups and within each group. Preliminary CCSG figures indicated a significantly ($p = 0.03$) lower overall relapse rate in those patients remaining on therapy (6.8% vs 26.3%, Table 1). However, more recent CCSG reports[15,16] have concluded that continuing therapy for five years rather than three does not significantly alter survival figures. The SWOG data, while not showing a significant reduction in the overall relapse rate for those treated longer, does, however, show a significantly ($p = 0.01$) lower rate of testicular relapse in males remaining on treatment (Table 1). It should be noted however, that SWOG reports a much higher rate of testicular relapses in long-term ALL survivors than any other series, for reasons which are unclear at this time. The therapeutic implications of the randomized CCSG-SWOG data must await larger patient numbers, longer followup times and perhaps another study with equality of patient populations and treatment protocols before any meaningful conclusions can be drawn.

In our study, 38/40 patients in complete continuous remission at three years of therapy were continued on treatment for an additional two years. Six (15%) relapsed during this period of continuing therapy and one died, in remission, from a chemotherapy-related infectious complication. Of the 33 patients still in complete continuous remission at five years and removed from therapy, only one (3%) has subsequently relapsed, bringing to 7/40 (17.5%) our total relapse rate in patients in complete continuous remission at three years (with treatment continuing for a median of 61 months). This figure is very similar to the mean relapse rate of 18.75% seen in the literature review of 752 patients, most of whom were treated for three years or less, thus indicating no added benefit to continuing treatment beyond three years.

continued over

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VIRGINIA AUTHORS

Sarcoidosis in Japanese and American Children. E. L. Kendig, Jr., MD, *Richmond,* and Y. Niitu, MD, *Sendai, Japan.*

A study of sarcoidosis among Japanese and American children suggests that there are more than the previously suspected number of children with asymptomatic sarcoidosis. The annual chest roentgenogram in Japanese school children detected the majority of these cases, while in the United States, where the annual chest roentgenogram is not required, the majority of children had sarcoidosis diagnosed through symptomatic complaints. Chest 77:514, 1980

Hypoglycemia and Unilateral Pulmonary Edema in a Newborn (Grand Rounds Series). K. W. Kerkering, MD, L. W. Robertson, MD, M. B. Kodroff, MD, D. G. Mueller, MD and B. V. Kirkpatrick, MD, *Richmond.*

A case of cardiomegaly with unilateral pulmonary edema is presented. The predisposing etiologies were severe hypoglycemia and a hypertonic glucose infusion into a branch of the left lower lobe pulmonary vein. Resolution resulted following maintenance of adequate glucose levels, vigorous cardiopulmonary support and repositioning of the catheter tip in the inferior vena cava. The pathophysiologic mechanism was direct endothelial injury with an osmotic diuresis. The role of the release of a vasoactive substance was postulated. Pediatr 65:326, 1980

Mini-Laparotomy for Bilateral Tubal Ligation in Lithotomy Position. William E. Byrd, MD, *Norfolk.*

Two hundred consecutive cases of mini-laparotomy for non-puerperal bilateral tubal ligation done by a single gynecologist with the patient in lithotomy position are presented here. Technical aspects of the procedure, some innovative and some old, are presented in an effort to demonstrate a simple, low-risk alternative to laparoscopic sterilization. South Med J 72:1554–1556, 1979

Penetrating Wounds of the Buttock. Kimball I. Maull, MD, J. William Snoddy, MD, and B. W. Haynes Jr., MD, *Richmond.*

Penetrating injuries of the buttock comprise a clinical syndrome largely ignored in the literature. Experience at the Medical College of Virginia with 15 consecutive patients having penetrating wounds of the buttock suggests that this injury produces a spectrum of injury from minor soft tissue damage to severe internal destruction and that, in most instances, aggressive surgical management is required. A review of this clinical experience forms the basis of this report. Surg: Gyn Obstetr 149:855–857, 1979

Suppression of ACTH by Cortisol in Dexamethasone-Nonsuppressible Cushing's Disease. Robert M. Carey, MD, *Charlottesville.*

The high-dose (8 mg per day) dexamethasone suppression test, as described originally by Liddle, has come to be accepted as one of the most reliable means of identifying Cushing's syndrome due to excessive pituitary ACTH secretion (Cushing's disease). However, a few cases of hypercortisolism with bilateral adrenocortical hyperplasia have been described with resistance to dexamethasone suppression, and this report describes a patient with Cushing's disease in whom the hypothalamic-pituitary-adrenal feedback mechanism failed to recognize dexamethasone as a suppressant glucocorticoid. N Eng J Med 302:275–279, 1980

Staphylococcal Ophthalmia Neonatorum and the Staphylococcal Scalded Skin Syndrome. Kenneth R. Fox, MD, *Arlington,* and Herbert S. Golomb, MD, *Washington.*

Ophthalmia neonatorum is usually a mild, catarrhal blepharoconjunctivitis. Staphylococcal organisms are a frequent cause of ophthalmia neonatorum, and occasionally they may cause serious ocular complications of the cornea, the globe itself, or the ocular adnexal structures. Another complication is the staphylococcal scalded skin syndrome, a generalized exfoliative dermatitis that frequently involves neonates and results from an initial focal staphylococcal infection. This report describes such a case. Am J Ophthalmol 88:1052–1055, 1979

Myringotomy: A Neglected Office Procedure. Richard H. Schwartz, MD, *Vienna.*

An office myringotomy is an easily performed, underused procedure which has definite clinical applications. The family physician should become familiar with the technique, its indications and its complications. Indications include otitis media with

concomitant meningitis, with mastoiditis or with cranial nerve involvement, and otitis media in an immunocompromised patient, in a neonate with signs of sepsis or in a very sick, toxic child. If the incision is made in the lower portion of the tympanic membrane, complications are rare. Am Fam Physician 20:102–108, 1980

Chronic Neck and Back Pain: A Reassessment of Usual Surgical Treatment. Charles A. Cohen, MD, H. F. Young, MD, J. R. Howell, MA, E. R. Griffith, MD, and D. P. Becker, MD, *Richmond.*

One hundred seventeen patients with chronic (noncancerous) back or neck pain had multidisciplinary evaluation in the Medical College of Virginia Pain Center and were followed up for one year. They ranged in age from 26 to 57 years. The 57 patients who had surgery had an average of 2.5 procedures (either laminectomies or back fusion) and only five of them returned to work. By contrast, 16 of 60 patients who had not had operation were able to secure employment after evaluation and treatment in the Pain Center. South Med J 73:40–42, 1980

Falciform Ligament: Varieties of Imaging Experience. Anthony J. Buschi, MD, A. Norman A. G. Brenbridge, MD, C. David Teates, MD, and Brian R. J. Williamson, MD, *Charlottesville.*

The falciform ligament is commonly imaged by various modalities available to most radiologists. When displayed, its diagnostic value is limited, but knowledge of its appearance as encountered on different examinations is useful, lest pathologic significance be applied to its appearance. South Med J 73:318–321, 1980

Methyldopa Inhibition of Suppressor-Lymphocyte Function. A Proposed Cause of Autoimmune Hemolytic Anemia. Howard H. Kirkland III, MD, Daniel N. Mohler, MD, and David A. Horwitz, MD, *Charlottesville.*

To test the hypothesis that methyldopa induces red-cell autoantibodies by inhibiting the activity of suppressor lymphocytes, we studied its effect on several immune functions. Methyldopa inhibited T-lymphocyte suppression of IgG production by peripheral-blood mononuclear cells stimulated by pokeweed mitogens. This effect occurred in isolated T cells incubated with methyldopa and in T cells obtained from patients taking methyldopa. In addition, the drug caused a 30 to 80 per cent reduction in the proliferative response of peripheral-blood mono-

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nuclear cells to mitogens in vitro, and this reduction primarily involved the activation of T lymphocytes. Methyldopa also caused a persistent elevation of intracellular lymphocyte cyclic AMP in vitro and in vivo. We postulate that methyldopa alters the immune system by causing a persistent increase in lymphocyte cyclic AMP, which inhibits suppressor T-cell function. These effects may lead to unregulated autoantibody production by B cells in some patients. N Engl J Med 302:825–832, 1980

Free Plasma DNA in Patients with Pulmonary Embolism. F. A. Barada, Jr., MD, Paul M. Suratt, MD, John S. Davis IV, MD, James N. Sipes, MD, Charles A. Castle, MD, Ronald P. Taylor, PhD, and Stephanie M. Godfrey, *Charlottesville.*

Plasma samples from 46 patients with suspected pulmonary embolism and 25 patients with suspected acute myocardial infarction were analyzed for DNA by counterimmunoelectrophoresis (CIE). Anti-DNA serum was obtained from a patient with systemic lupus erythematosus who had a high titer of anti-DNA antibodies. Seven of eight patients (88%) with high probability of pulmonary embolism by lung scan criteria had free DNA in their plasma. Six of 21 patients (29%) with low probability scans for pulmonary embolism also had DNA in their plasma. None of the eight patients with normal scans and only one of 13 patients with myocardial infarction had DNA in their plasma. Detection of plasma DNA by CIE is a rapid and simple method of screening for pulmonary embolism. South Med J 73:345, 1980

***Serratia marcescens* Bacteremia from Contaminated Pressure Traducers.** Leigh G. Donowitz, MD, Frederick J. Marsik, PhD, John W. Hoyt, MD, and Richard P. Wenzel, MD, *Charlottesville.*

Primary *Serratia marcescens* bacteremia developed in 17 patients in an intensive care unit after exposure to pressure monitoring devices. A study showed that all of the transducer heads were contaminated with *S marcescens*, and prospective culturing of 110 pressure monitoring lines disclosed a 24% rate of contamination with the same organism. Hand contamination occurs at the time the equipment is initially assembled; in five of eight trials, transmission was shown experimentally to occur by direct inoculation of open ports. Routine disinfection of the transducer heads with glutaraldehyde not only effectively decontaminated all pressure monitoring lines in use, but also controlled the outbreak. JAMA 242:1749–1751, 1979

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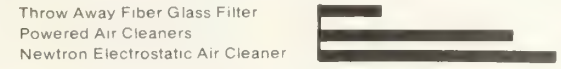
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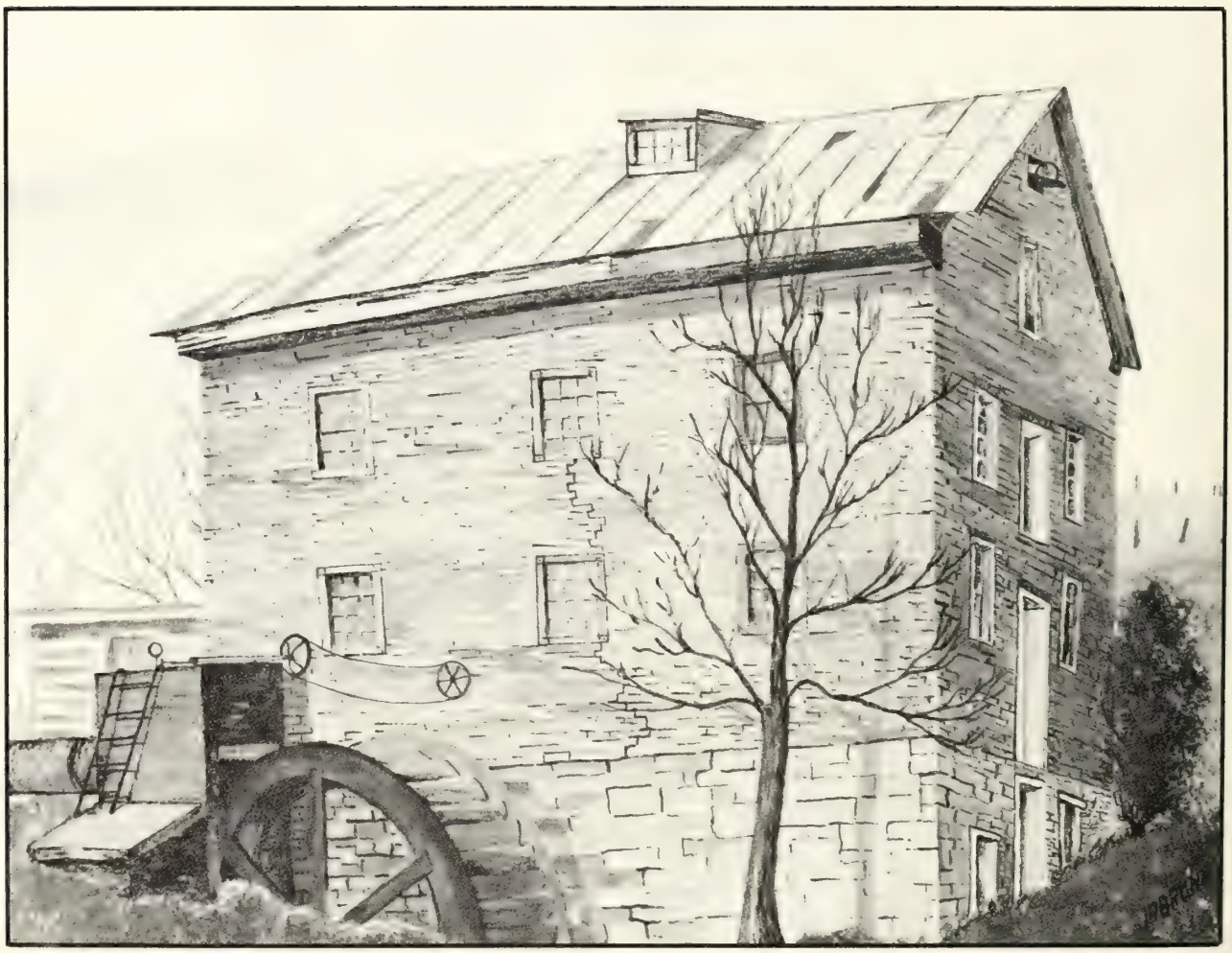

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VIRGINIA MEDICAL

EDITORIAL

Does your generosity need exercising? Start here.

THE MEDICAL PROFESSIONAL is being subjected to heavy criticism, most of it unjustified. There is one area in which we may not be blameless.

Before the advent of Medicaid, Medicare and all the other third-party payers, physicians performed many services with no expectation of monetary return. These services constituted the physician's contribution to the community and to the Commonwealth. For all practical purposes, this situation no longer exists, but many physicians have made no adjustment in their approach to giving; the medical profession is not regarded as a generous contributor to community causes.

Of all the programs that merit the support of the physicians in Virginia, high on the list must be that of the Virginia Council on Health and Medical Care. This voluntary, privately supported organization, headed so ably by Edgar J. Fisher, Jr., has been functioning effectively since 1946.

The Virginia Council is probably best known to the medical profession for its Physician Referral Service, a clearing house to bring together areas of the state needing physicians with physicians who are looking for a place to practice. Through this aggressive and efficient service 1,275 physicians have found practice locations in 243 communities throughout the Commonwealth.

In addition, the Virignia Council also provides referral services for dentists, occupational therapists, dental hygienists, a Health Careers Program, sponsors conferences on the handicapped child and co-sponsors an annual nutrition forum with Thalhimer's Department Store. On request, the Council arranges and co-sponsors conferences on specific topics important to health and medical care. Since 1964, the council has provided a forum for the executives of the statewide voluntary health organizations to come together quarterly for informal programs of interest and concern to all.

Members of the staff of the Virginia Council answer a wide range of mail and telephone inquiries relating to health and medical care and the availability and location of facilities and sources. These inquiries may originate out of state as well as from organizations and individuals in the state.

Monies from the Virginia Council 1982 budget of $272,689 came from 926 different donors, derived primarily from the following sources: business, industrial and banking concerns; health professional organizations; hospitals, nursing homes and medical clinics; foundations and trusts; and individuals. Two-hundred-fifty-one individual physicians contributed a total of $14,491.50.

In a letter written in 1982, Dr.Wyndham B. Blanton, Jr., then president of the Virginia Council on Health and Medical Care, requested contributions from 320 physicians placed by the Council. Only 20 (6%) responded, with a total contribution of $1,575.

The Medical Society of Virginia and other state medical organizations have always given strong support to the Council. As individual physicians, we must do more to support this great organization that contributes so much to medicine and medical care in Virginia. I have made a small annual contribution as long as I can remember, but it hasn't been large enough. As I complete this editorial, I am sending more. I hope all other Virginia physicians will do likewise.

EDWIN L. KENDIG, JR., MD

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VIRGINIA MEDICAL OBITUARY

Memoir of Algie C. Davis 1905–1982

By Duvahl Hull, MD, Robert E. Paine, Jr., MD, and James G. Snead, MD

Dr. Algernon C. Davis, age 77, died August 7, 1982, at his home in Roanoke.

Algie was born in Riner, Virginia, on June 6, 1905, one of five sons of Dr. John Gibson Davis and Zora Lucas Davis. All five became physicians and members of the Roanoke Academy of Medicine. In addition to Dr. Algie Davis the brothers were the late Dr. Hal Davis, the late Dr. Paul Davis, the late Dr. John Gibson Davis, and Dr. Fred F. Davis, who alone survives.

Dr. Algie Davis grew up in Roanoke and was educated in the public schools. He was graduated from Roanoke College and enrolled in the Medical College of Virginia, graduating in 1929. He and his four brothers practiced in Roanoke.

Dr. Davis was an avid tennis player. In the 1927 city tournament finals, he defeated his older brother, Gib, and was the first official tennis champion in Roanoke. With his younger brother, Hal, he continued to play competitive tennis until 1935.

He served in the Army Medical Corps during World War II and was in the Pacific Theater of operations, stationed in the Army hospital on Siapan. Following discharge from military service, he resumed his private practice of obstetrics in Roanoke.

A warm and compassionate physician, he was concerned not only with the physical needs of his patients, but with their emotional needs as well. He endeared himself to his many patients as well as to his colleagues. His gentle manner was his hallmark.

He was proud owner of a farm in the beautiful Catawba Valley, where he loved working with his horses and raising corn. His seven grandchildren nicknamed him "Grandcorn." He had one of the first radio-page systems in the Roanoke Valley so that he would be reached when off-duty. He was a member of the First Baptist Church of Roanoke.

His lovely wife, Flo, preceded him in death recently. He is survived by his brother Fred; by two sons who are practicing physicians in Roanoke, Dr. William V. Davis and Dr. A. Gibson Davis; and by seven grandchildren.

Algie will be missed by his many patients, his colleagues and his family.

Prepared and published at the request of the Roanoke Academy of Medicine.

Joseph D. Casolaro, MD

Dr. Joseph Daniel Casolaro, Arlington obstetrician and gynecologist, died of leukemia at age 60 on April 30, 1982, at the University of Miami Hospital in Miami. He lived in Waterford, Virginia, and had gone to Miami for treatment.

A native of New York City, Dr. Casolaro was graduated from Fordham University, Bronx, New York, and earned his medical degree at the Long Island College of Medicine. Following service in the Army during World War II, he established his practice in Northern Virginia and was also a clinical professor at the Georgetown University School of Medicine until his retirement in 1975. For 30 years a member of The Medical Society of Virginia, Dr. Casolaro was also a member of the Northern Virginia Medical Society and the Fairfax County Medical Society.

A son, Dr. Anthony Casolaro, practices medicine in Atlanta.

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WHO'S WHO

To **Dr. Emil S. Sayegh,** who was born in Cairo and came to Virginia in 1957, was given Portsmouth's First Citizen Award in recognition of his "untiring work to improve the quality of health care" in the Tidewater area.

A past president of the Portsmouth Academy of Medicine, Dr. Sayegh has been chairman of the Portsmouth Museum and Fine Arts Commission and has served on the boards of many civic enterprises, including the Tidewater Kidney Foundation and the Hampton Roads Foreign Affairs Council. In the late 1960s he established the first kidney dialysis unit in Tidewater, offering his urological services free to patients who needed the unit, and he has been chief of staff at both Portsmouth General and Maryview Hospitals.

At 70, Dr. Sayegh has curtailed neither his private practice nor his association with Eastern Virginia Medical School, where he is now associate professor of urology.

Dr. Sayegh was educated in British and American schools. When he left Egypt, because of political differences with the Nasser regime, he enlisted in the United States Navy and was commissioned a captain and assigned to the Portsmouth Regional Medical Center. He entered private practice in Portsmouth in 1965.

Dr. James O. Willie. Norfolk, received the 1982 Health Service Award from the Society for the Aid of Sickle Cell Anemia at its 10th anniversary awards dinner.

For his many years of faithful service to Georgetown University, **Dr. Albert J. Orlosky,** Arlington pediatrician, was given the university's Vicennial Award. A reception and dinner marked the presentation.

The Rockingham County Medical Society has elected these new officers: **Dr. Paul R. Yoder,** president; **Dr. Richard M. Senfield,** vice president; **Dr. Frederick L. Fox,** secretary; and **Dr. Regis C. Kohring,** treasurer. All practice in Harrisonburg.

Gov. Charles S. Robb has appointed **Dr. Edgar Newman Weaver,** Roanoke, to the board of visitors of the University of Virginia.

Dr. John A. Owen, Jr., is the newly elected president of the Albemarle County Medical Society. Elected to serve with him were **Dr. William A. Orr,** vice president and president elect; and **Dr. George R. Hanna,** secretary-treasurer. All practice in Charlottesville.

Tapped for a 1983 Distinguished Service Award by the Fredericksburg Jaycees was **Dr. Robert A. Kravetz,** Fredericksburg anesthesiologist, who is president of the area's Emergency Medical Services Council and medical advisor of the Fredericksburg Rescue Squad, and led a successful local drive to acquire telemetry equipment for the rescue unit.

Dr. Walter R. Zolkiwsky, Sterling, is the new president of the Loudoun County Medical Society, and **Dr. Thomas J. Gates,** Leesburg, is the new secretary.

The Accomack County Medical Society has elected **Dr. Walter A. Eskridge,** Parksley, as its new president and **Dr. Parker C. Dooley,** Onley, as vice president, and has reelected as secretary **Dr. Belle D. Fears** of Accomac.

Cairo's loss was Portsmouth's gain: First Citizen Sayegh.
Photo by Mort Fryman for the Norfolk Virginian-Pilot

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References: 1. Kales A *et al: J Clin Pharmacol 17*:207-213, Apr 1977 and data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Kales A: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **3.** Zimmerman AM: *Curr Ther Res 13*:18-22, Jan 1971. **4.** Kales A *et al: JAMA 241*:1692-1695, Apr 20, 1979. **5.** Kales A, Scharf MB, Kales JD: *Science 201*:1039-1041, Sep 15, 1978. **6.** Kales A *et al: Clin Pharmacol Ther 19*:576-583, May 1976. **7.** Kales A, Kales JD: *Pharmacol Physicians 4*:1-6, Sep 1970. **8.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **9.** Dement WC *et al: Behav Med 5*:25-31, Oct 1978. **10.** Vogel GW: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **11.** Karacan I, Williams RL, Smith JR: The sleep laboratory in the investigation of sleep and sleep disturbances. Scientific exhibit at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971. **12.** Pollak CP, McGregor PA, Weitzman ED: The effects of flurazepam on daytime sleep after acute sleep-wake cycle reversal. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, June 30-July 4, 1975. **13.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ.

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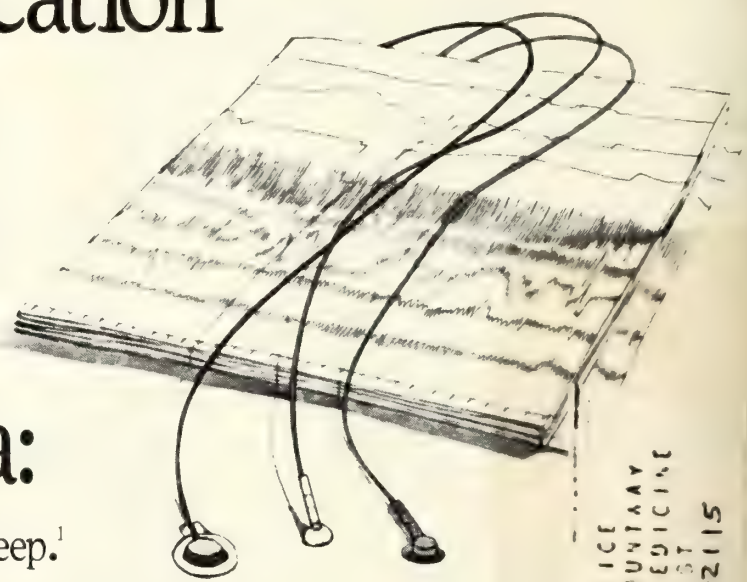
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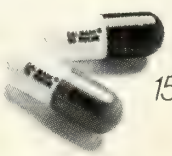

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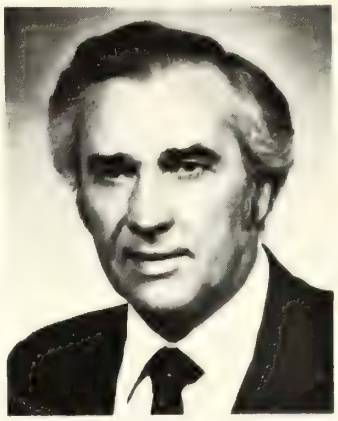

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From the VAMEDNE

The DRGs are coming, the DRGs are coming

By Carl L. Shires

FORESHORTENED HOSPITAL STAYS and fewer lab tests for Virginia's Medicare patients are the most immediate results that will come from the latest of the government-imposed health care plans, diagnostic-related groups, or DRGs, the prospective pricing system to be phased into effect beginning October 1.

Over the long haul, there will likely be less jingle in the jeans of Virginia's physicians, and a reduction of beds and a trend toward treatment specialization in Virginia's hospitals.

DRGs, yet another in the ever-proliferating acronymns affecting health care professionals, mean that hospitals face a future wherein they will be under the gun of a law involving Medicare reimbursement according to diagnoses in 467 categories at rates established by the Secretary of Health and Human Services.

"Get 'em in and get 'em out and don't do too much testing while they're there." That could become standard treatment policy if hospitals are to realize a profit from their Medicare patients, who now account for approximately 40% of hospitals' income.

The phase-in period is four years. Hospitals whose fiscal year begins October 1 will have but a month to ponder the final DRG rules, regulations and rates, which are to be formally proclaimed September 1.

Already, however, there are those who think that soon some other Congress-approved health care plan will come down the regulatory road to replace DRGs. One of them is Dr. Ronald K. Davis, Richmond vascular surgeon and president of the Richmond Academy of Medicine. Another is Richard O'Hallaron, veteran hospital administrator in Richmond.

Said Dr. Davis: "Nobody in their right minds thinks this is going to work, that it's going to do any good in reducing health care costs. There will be something to replace it fairly quickly." Said O'Hallaron, who as executive president is the chief administrator of St. Mary's Hospital in Richmond: "I think DRGs are temporary; I'd give them three to four years at the most."

There is justification for their predictions. Only a year ago, TEFRA, which stands for the Tax Equity and Fiscal Responsibility Act, was the government's acronym of choice for Medicare reimbursement. But TEFRA, which is based on retrospective payment, is to be phased out as the DRG prospective payment is phased in and will be on the discard heap by late 1986.

As a whole, hospital administrators do not seem overwhelmingly dismayed with the notion of DRGs. The American Hospital Administration approves the new concept—if for no other reason than that the AHA feels it is more acceptable than TEFRA.

Analysis of a summary of views elicited for VIRGINIA MEDICAL indicates that most health care professionals think hospitals can do well financially under DRGs if they run an efficient hospital ship, don't over-invest in cosmetic equipment,

For more on DRGs, see Dr. Brown's editorial on page 505.

get Medicare patients in and out in quick order, and discourage effectively the use of what they term "unnecessary" diagnostic tests.

"Unnecessary" in that context is a word that runs into different interpretations among physicians, hospital administrators and third-party insurers. "In all I've read and heard of DRGs, I've not been hearing or reading of any emphasis on quality care," noted Dr. Davis. "And who," he asked rhetorically, "is to decide what is quality care if it is not the attending doctor?"

"Education." That's the catch word when non-physician sources interviewed by VIRGINIA MEDICAL talk of how they propose to persuade physician-staffers to cooperate in cost cutting for a Medicare patient under DRGs. But some of them—and they did not want to be quoted by name on that subject—talked more darkly of confrontation if hospital stays and tests were running above the norm. And a few even mentioned the more drastic possibility of removal from hospital staffs for non-cooperation.

In Washington, a government man, F. Lynn May, said that for DRGs to operate successfully, "physicians will have to play a very important role. They will have to understand and cooperate. . . Successful hospitals under the plan will be the ones that effect that understanding and cooperation." May, who bears the formidable title of associate administrator, External Affairs Division, Health Care Financing Administration, Department of Health and Human Services, is scheduled to speak on Saturday, November 5, at The Medical Society of Virginia's annual meeting at the Homestead.

Are physicians' fees next in line for Medicare reimbursement on the same diagnosis-related basis? May declined to speculate on that question, but he acknowledged it "might be a fair reading of a trend." Indeed, Congress already has ordained that a study of that possibility be completed by next year.

And in Kansas, on January 1, 1984, that state's Blue Cross Blue Shield will begin paying both contracting hospitals and participating physicians on the basis of diagnostic-related groups. Multiple insurers throughout the country have been studying the Kansas plan with considerable interest. Under the Kansas arrangement, affected hospitals and physicians will be guaranteed full reinbursement up to maximum established rates. Other doctors and hospitals will bill patients directly and the patients will be reimbursed at the established rates. All BC/BS patients in Kansas—not just Medicare ones—will be affected.

On DRGs in Virginia, Dr. Richardson Grinnan, vice president of medical affairs for the Virginia Blues, thinks doctors will determine the success or failure of DRGs. "Physicians will be the key," he said.

He had this observation from the Blues' point of view, however: "There certainly is the potential to cut the costs of Medicare patient treatment, but that could result in a cost shift to third-party payers."

Among his other observations: The "strong" possibility for specialization in patient treatment by hospitals and the closing of some hospital beds in Virginia.

He was joined in both those observations by Stuart D. Ogren, executive vice president of the Virginia Hospital Association. Of the 31,357 hospital beds in the state, said Ogren, "there may now exist a surplus of 1,800 beds."

Ogren predicted also there will be an increased emphasis on deductibles in hospital insurance and a probable rise in other plans for cost-cutting, such as HMOs and PPOs. "I think we'll be seeing more people popping glycerin instead of having bypasses, and more people bearing with the discomforts of such things as hemorrhoids and ruptures," he put it colorfully. He qualified his bed-closing predictions, however. "While there may be some, the offsetting factors are the aging population and the increased quality of life expectations.

Dr. Davis offered the most point-blank prediction of hospital-bed closings as a result of DRGs. "There will be hospitals going out of business and wings closed," he said, "maybe up to 200 beds in the Richmond area alone."

O'Hallaron, from the 369-bed St. Mary's Hospital, said one major fault in the DRG proposal is that there is "no provision in the reimbursement plan that gives credit for indigent patients. That's a leverage against hospitals trying to take care of the poor and we try to do that

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here." Despite that, though, he said he feels "we'll do well."

Despite no provisions for indigent care, the proposed DRG plan does have some adjustments intended to ease the extra financial loads borne by sole community providers and by teaching and long-term-care hospitals. Initially, there will be adjustments also for urban-rural location differences and the ensuing disparities in labor costs.

Thomas W. Leggett, executive director of Community Memorial Hospital in South Hill, represents one of those sole-provider, rural hospitals.

"There are just too many 'ifs' at this time, before they give us the final details, to make smart predictions," he said, "but we're going to have our problems. We serve three rural Virginia counties and parts of North Carolina, and lots of our patients live far away and some of the roads are bad in the winter. Some of those patients may have to stay an extra day and we'd lose money on that. Also I don't know what's going to be in the formula—if anything—to provide for capital improvement and return on equity. We've got a lot of retirement people in this area and about 52% of our patients are on Medicare.

"I've got mixed emotions, as do most of my administrative colleagues at similar hospitals." Leggett is chairman of the Virginia Hospital Association's committee on small and rural hospitals.

Miles Lash, for 13 years the executive director of the Medical College of Virginia Hospitals, is withholding final predictions on DRGs till he sees the final plan in September but has reservations.

"It does not take into account the severity of illnesses—such things as our burn unit and pediatric intensive care. And teaching hospitals historically get heavy loads of indigent and acutely ill patients."

Each of the hospital administrators questioned talked of the importance, under DRGs, of medical records personnel. It will be they who will ultimately determine under which of the 467 codes the hospitals will be paid. On the coding can depend the profit or the loss. There will be penalties for willful miscoding, said May.

A colleague in May's department, Daniel Bourque, deputy administrator of the Health Care Financing Administration, said at a meeting of the American Academy of Family Physicians recently that the coding will not be all that formidable. Of the 467 categories of diagnosis, he said, only about 350 apply to Medicare payments, and 30 will account for more than half of all reimbursements.

In addition to the impact of DRGs on the relationship of hospitals and physicians, there is another aspect of importance, that of the relationship between physicians and patients. Writing in the May issue of the *Journal of the Florida Medical Association,* Dr. Henry L. Harrell, Jr., expressed it this way:

"We will no longer be able to enjoy the luxury of ordering all the tests that might conceivably be of help. There will be pressure to do only what is necessary. We will no longer be able to keep the patient an extra day or two because of a special problem at home. . . It is true that intensity of care and length of stay have both been abused in the past when all costs were reimbursed and there was no incentive to avoid their abuse. It is also true, however, that decisions as to what is appropriate intensity of care can be extremely difficult, and length of stay can sometimes be legitimately argued on the basis of important personal concerns not directly related to the patient's diagnosis. It is certainly appropriate for a physician to risk doing a little too much for his patients in an attempt to avoid the risk of doing too little. . . ."

VIRGINIA MEDICAL

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*Shils ME, Randall HT: Diet and nutrition in the care of the surgical patient, chap. 36, in *Modern Nutrition in Health and Disease*, edited by Goodhart RS, Shils ME; Philadelphia, Lea & Febiger, 1980, p. 1084.

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LETTERS

Alexandria's cats slated for rabies vaccination

In the article on rabies vaccine in the June issue of VIRGINIA MEDICAL,[1] a statement was made that five counties in Northern Virginia, acting on Dr. Kenley's recommendation, passed local ordinances requiring vaccination of cats against rabies.

Would you amend that statement to read "five counties and one city"? The City Council of Alexandria, in February of this year, amended the ordinance requiring vaccination of dogs to include vaccination of cats.

Anne J. Albertson, MD

Director, Alexandria Health Department
517 North Saint Asoph Street
Alexandria VA 22314

1. Shepard GH et al: New vaccine at the ready in rabies risk. Va Med 110: 340–342, 1983

"We must work together or surely we will fail"

In the March issue VIRGINIA MEDICAL carries a speech by Dr. Ronald K. Davis delivered before the Richmond Academy of Medicine when he was inaugurated as its president this year.[1] Dr. Davis comments that "the number one reason why health care costs have risen faster than all other goods and services, except energy and government, is the government itself." Further, he equates PSROs with government and makes reference to their ineffectiveness and high cost of maintenance.

The Shenandoah Professional Standards Review Organization, through its board of directors and working committees, embodies the physician leadership of the Charlottesville community and has made every effort to control costs and suggest quality control improvements in the delivery of health care.

We have consistently "saved" several times our expenditures. I have testified before Congress and presented evidence supporting our effectiveness in muting over-utilization. On the matter of quality we

are constantly evaluating audits of hospitals and suggesting improvements as well as performing audits where appropriate. One can argue that all we have accomplished is the shifting of costs and that the inflationary trends have continued unabated. This, however, does not make us any less efficient. It does say that something is wrong with the system of which we are a part.

All of us, in both the private and public sectors, are party to a system of reimbursement that provides an economic incentive for over-utilization of physician and hospital services. We are also caught in an industry undergoing a technological revolution both in terms of unprecedented capital cost and a concomitant dehumanization which, among other things, has brought about a discontent in both patient and physician, culminating in increases in malpractice suits.

We are facing a national crisis. In 1982 the total cost of medical care reached 10% of our gross national product. By 1985 it will reach 11%. Medicare and Medicaid represent 45% of the total annual medical budget and, if the present trend continues, authorities say that figure will increase to 88% in five years' time and that by 1988 the Medicare trust fund will be depleted.

The costs of medical care, both private and public, are rising at 14% annually. The private sector can anticipate an even faster rise as we come to grips with the public sector.

Congress recently passed a system of reimbursement for Medicare that will provide an economic incentive for under-utilization called "prospective payment." This new system has its problems. It is extremely complex.

The decade ahead will be most difficult and trying. We must work together or surely we will fail.

Henry B. Hunt, Jr.

Executive Director,
Shenandoah Professional Standards Review Foundation
716 West Rio Road, Charlottesville VA 22901

1. Davis RK: "Why do doctors complain about Medicare?" Va Med 110:192–193, 1983

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And Mr. Reagan rang down the curtain

Words by Raymond S. Brown, MD
Photographs by Arthur A. Kirk, MD

THE 138th annual meeting of the American Medical Association in Chicago drew 351 delegates, including this Medical Society of Virginia delegation: Mike Puzak, our capable and respected leader; Bill Hotchkiss, Pete Carmines, John Martin, Len Weyl, Bill Hagood, Arthur Kirk, Charlie Caravati, Percy Wootton, Chris Alexander, Barrie Cook, Harold Williams, and myself.

We joined in considering 149 resolutions and 70 board and council reports, and helped elect new officers and council members.

An exciting development occurred prior to the opening of this session, which was June 18–23. This was the formation of the new Medical Staff Section. Nearly 700 representatives from 500 hospitals over the country showed up for this organizational meeting. Their interest and commitment to American medicine was evidenced by their wide-ranging discussions and many resolutions, eight of which were acted on by the House of Delegates.

Our days began at 6 AM with working breakfasts at which we discussed resolutions and reports and met candidates for office. We attended the House of Delegates meetings in a body, but for the reference committee hearings we split up into small groups, so that all nine reference committees would be adequately covered.

The AMA's reference committees, like those of The Medical Society of Virginia, are conducted in an open and unhurried fashion. All AMA members have the privilege of the floor, and anyone with a legitimate interest in the topic under discussion can easily get the privilege. The debate is often hot and heavy. The reference committee assimilates the debate; previous AMA actions and reports relating to the topic; information from staff, trustees, and, in this day and time, the AMA Legal Department. From all of this, the committee prepares a report for the House of Delegates, where it is acted upon.

At this meeting, the resolutions ranged from one requesting a smoke-free environment by the year 2000 to one requesting a fixed indemnity system of reimbursement by all third-party payers. There were 27 resolutions pertaining to the Joint Commission of Accreditation of Hospitals; the delegates' action on these resolutions essentially protects the

◀ **Dressed for dinner: Dr. and Mrs. William S. Hotchkiss (left) and Dr. and Mrs. Michael A. Puzak. Dr. Hotchkiss, who is AMA secretary-treasurer, was elected to a second post as vice chairman of the Board of Trustees. Dr. Puzak is chairman of The Medical Society of Virginia's AMA delegation.**

▲ **Recess on the House floor: Dr. F. Ashton Carmines (left) and Dr. William J. Hagood, Jr.**

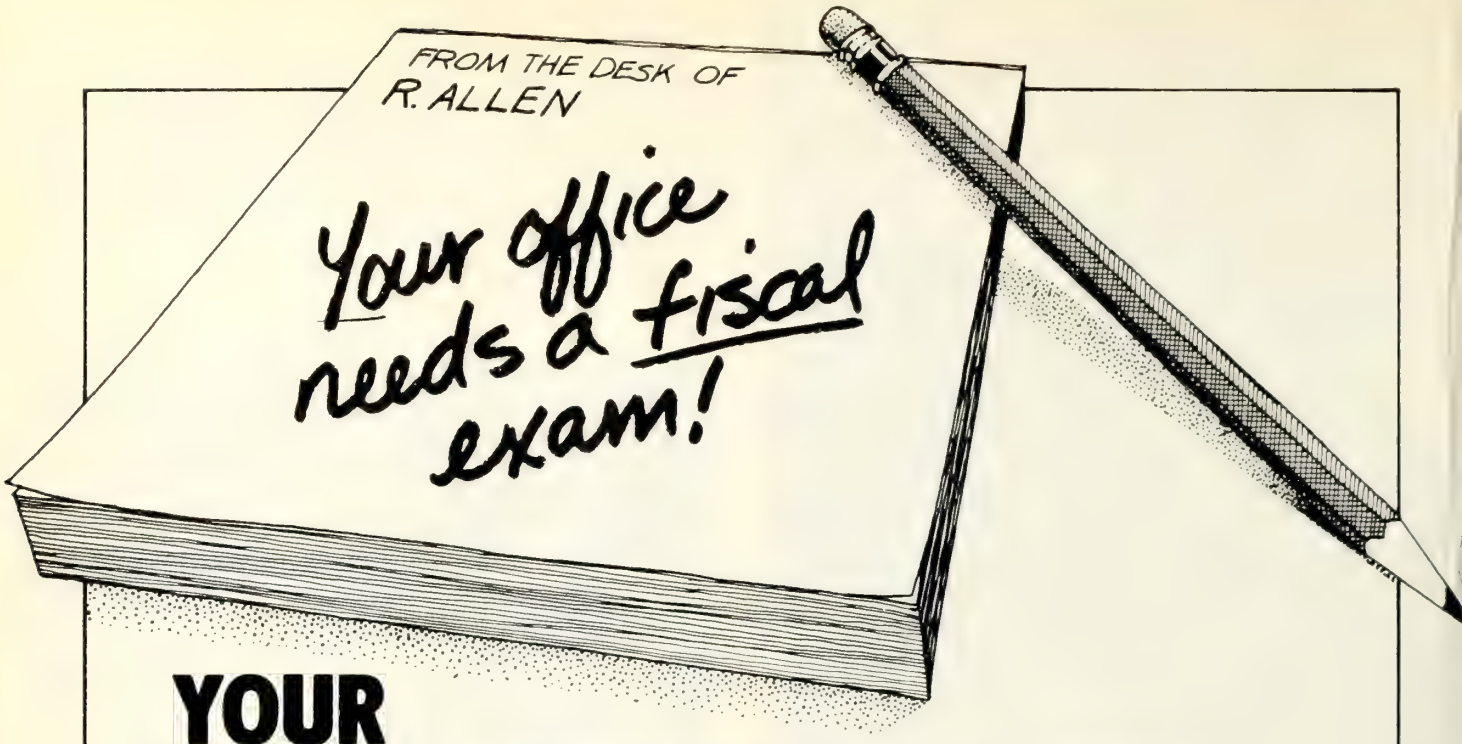
medical staff from dilution by practitioners other than licensed MDs or doctors of osteopathy.

Difficult issues are raised by some resolutions and reports, and decisions of the House frequently run counter to the short-term interest of many of our constituents but are made in the best long-term interest of physicians.

At the conclusion of the House's agenda on the last day, there was a visit by the President, Mr. Ronald Reagan, who gave a friendly, humorous speech that wound up in a serious vein with the request that physicians hold the line on costs by accepting a one-year freeze on Medicare reimbursements.

Since the media is apt to pick up unusual items in the resolutions, news reports of these meetings often have little relation to the importance of the items discussed. This year, however, there seemed to be fair and accurate coverage of two newsworthy topics: a resolution recommending a more scientific approach to dioxin and the request that the Council on Scientific Affairs prepare frequent reports on acquired immune deficiency syndrome.

▲ **Breakfast meeting: Dr. Harold L. Williams, MSV President (left), Dr. Raymond S. Brown (standing), and Dr. Charles M. Caravati.**



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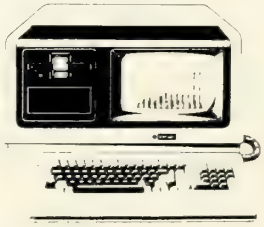

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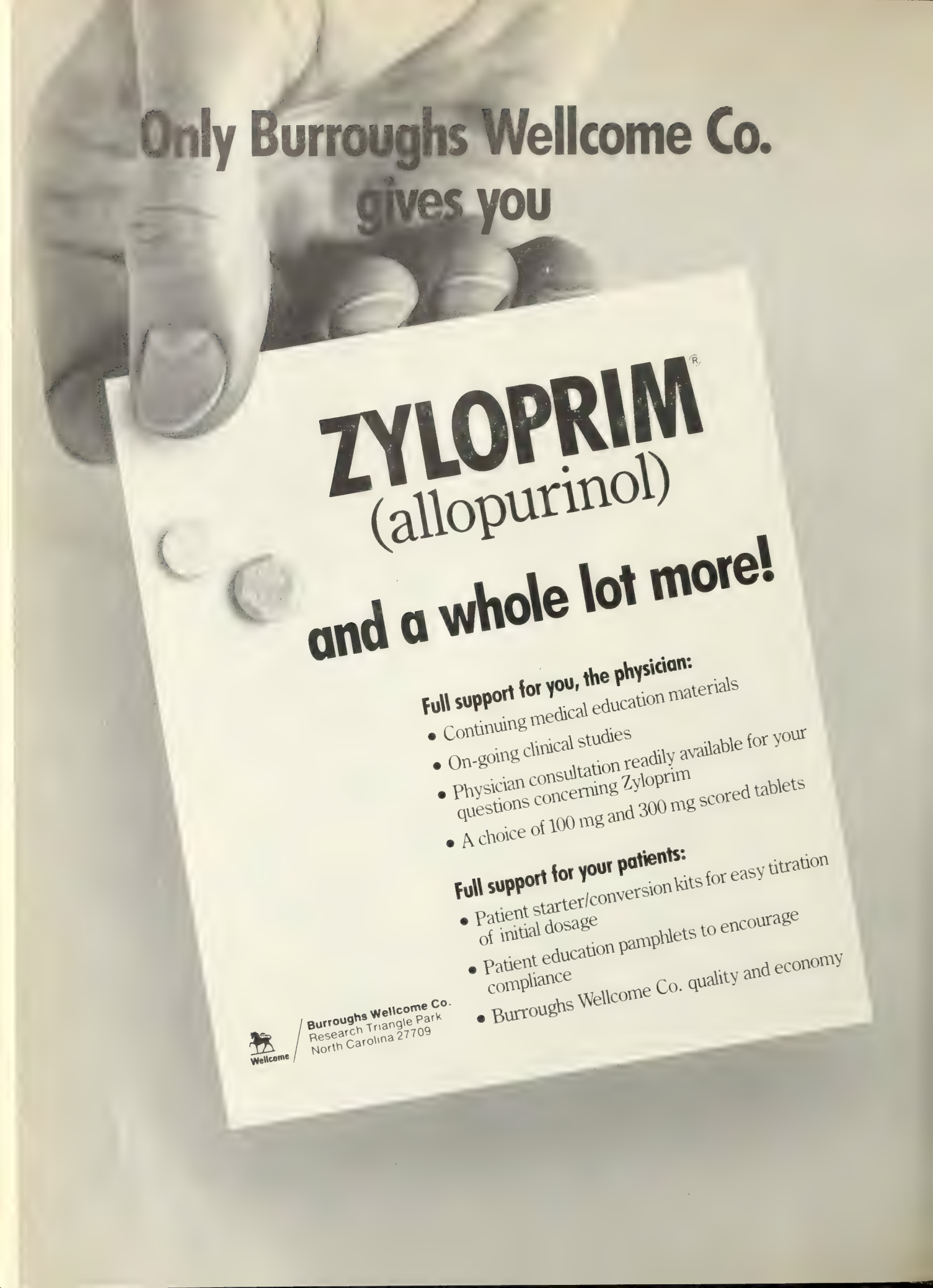
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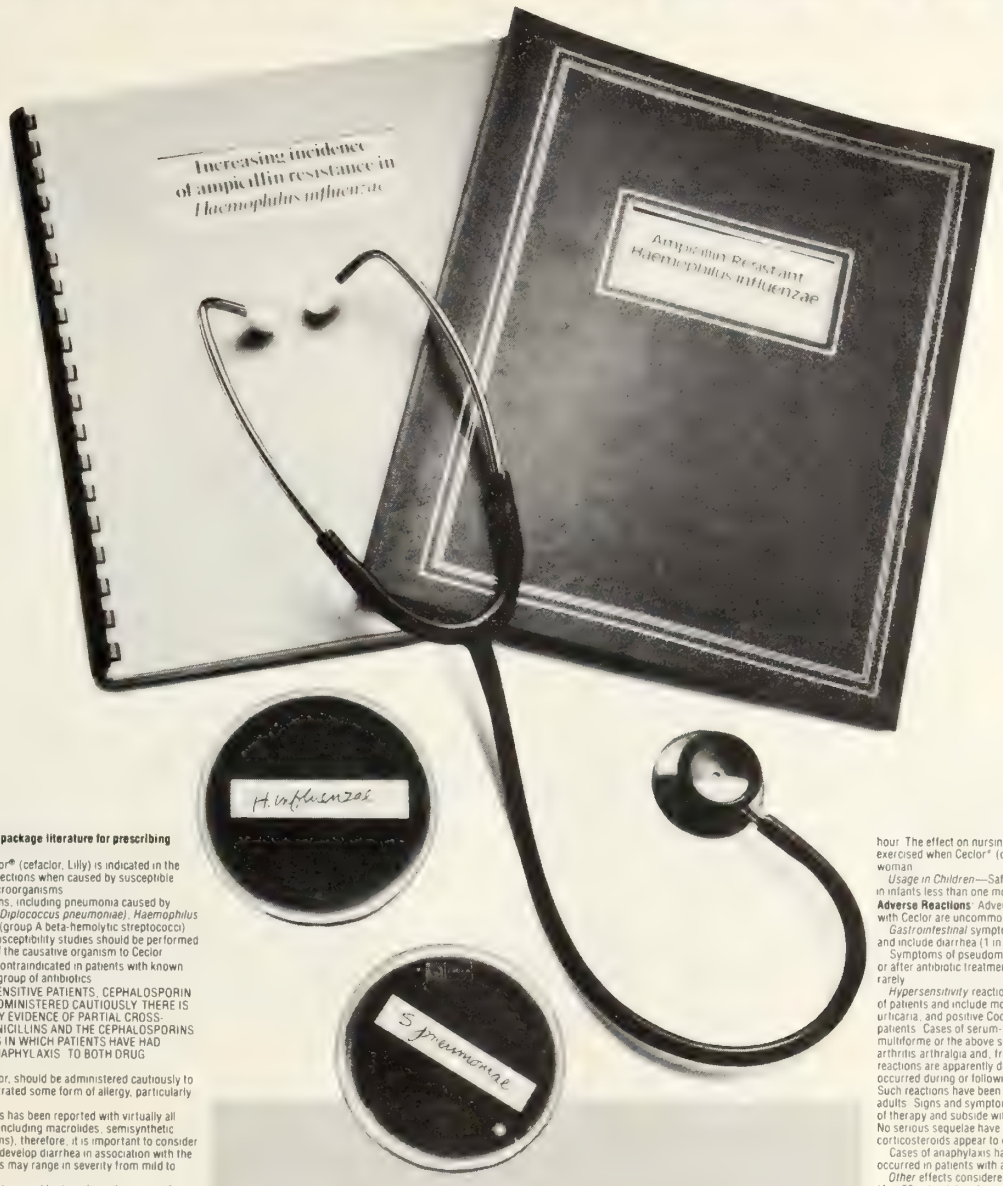
An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

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Brief Summary. Consult the package literature for prescribing information

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out

Precautions: *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy—*Pregnancy Category B*—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor* (cefaclor, Lilly) is administered to a nursing woman

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70)

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients)

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40)

Hematopoietic—Transient fluctuations in leukocyte count predominantly lymphocytosis occurring in infants and young children (1 in 40)

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200)

[061782R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information

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September 8-10
14th Annual Anesthesiology Symposium (Portsmouth Naval Hospital/Association of Military Surgeons of US), Omni Hotel, *Norfolk*. (804) 398-5125.

September 11-15
Clinical Skills Workshop and Review (Bowman Gray School of Medicine), *Winston-Salem, North Carolina*. 31 hrs., Cat I. Fee: $360. Division of CME, 300 Hawthorne Rd., Winston-Salem NC 27103.

September 15-16
Advances in Diagnostic Ultrasound (Johns Hopkins University), *Baltimore*. 16 credit hrs. Julia C. Burbridge, Office of CME, 720 Rutland Ave., Baltimore MD 21201, (301) 955-8450.

September 15
Anti-coagulant, Thrombolytic and Anti-platelet Therapy (Roanoke Memorial Hospital), *Roanoke*. Charles L. Crockett, MD, Roanoke Memorial Hospital, Roanoke VA 24033, (703) 981-7228.

September 15
10th Annual Bishop John J. Russell Lecture: "Nuclear Magnetic Resonance, a Non-Invasive Imaging Technique" (Peter L. Davis, MD, San Francisco), St. Mary's Hospital, *Richmond*. One hr. Cat. I. Edwin L. Kendig, Jr., MD, (804) 281-8441.

September 19-21
National Symposium on Colorectal Tumors (George Washington University), *Washington, DC*. Cat I credits. Fee. $395. Greg P. Thomas, Office of CME, 2300 K St., NW, Washington DC 20037, (202) 676-4285.

September 20
Current Concepts in Ophthalmology (University of Maryland), *Baltimore*. Michael E. Wessely, Program of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

September 22-23
6th Annual Conference on Aging and Health (Veterans Administration Medical Center/University of Virginia), *Salem, Virginia*. 10 hrs. Cat. I. Fee, $75. Frank Brochu, MD, Veterans Administration Medical Center, Salem VA 24153.

September 23-24
Partial Mechanical Support of the Failing Heart (University of Maryland), *Baltimore*. Michael E. Wessely, Program of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

September 25-28
Radiology Update (University of Virginia), *Charlottesville*. 20 credit hrs. Fee: $350. T. E. Keats, MD, Box 170, UVa Medical Center, Charlottesville VA 22908, (804) 924-2781.

September 26-30
Basic Practices for the Novice Ultrasonographer (Johns Hopkins University), *Baltimore*. 40 credit hrs. Fee: $400. Office of CME, 720 Rutland Ave., Baltimore MD 21201, (301) 955-8450.

September 27-29
Treatment of Hyperglyceridemia (National Institutes of Health), *Bethesda, Maryland*. Mrs. Peter Murphy, 2115 E. Jefferson St., Suite 401, Rockville MD 20852, (301) 468-6555.

September 29-November 3
Selected Topics in Family Practice (University of Maryland), *Baltimore*. 6 successive Thursday evenings. Michael E. Wessely, Program of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

September 30
Cardio-Pulmonary Diseases: Recent Advances (University of Maryland), *Baltimore*. Michael E. Wessely, Program of CME, 10 S. Pine St., Baltimore MD, 21201, (301) 528-3956.

September 30-October 1
Update in Rheumatology (Arthritis Foundation, Maryland Chapter), *Baltimore*. 9 hrs. Cat. I. Karen Goodman, 12 West 25th St., Baltimore MD 21218, (301) 366-0923.

October 6-8
Topics in Gastroenterology and Liver Disease (Johns Hop-

kins University), *Baltimore*. 21 hrs. Cat. I. Fee, $295. Noreen Javornik, Office of CME 720 Rutland Ave., Baltimore MD, 21205, (301) 955-6046.

October 7
Selected Topics in Pediatrics (Eastern Virginia Medical School/Children's Hospital of the King's Daughters), *Norfolk*. Cat. I credits. Jean E. Shelton, MD, 800 W. Olney Rd., Norfolk VA 23507, (804) 628-3798.

October 10-14
25th Annual Emil Novak Memorial Course in Clinical Gynecological Pathology, Obstetrics and Genetics (Johns Hopkins University), *Baltimore*. Carlita Kearney, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

October 12-14
Topics in Ambulatory Medicine (Johns Hopkins University/Baltimore City Hospital), *Baltimore*. 20 hrs. Cat. I. Fee, $275/MDs, $150/residents. Susan Bavaro, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-6046.

October 13-15
Child Neurology Society, Annual Meeting, *Williamsburg*. National Office, Box 486, Minneapolis MN 55455.

October 13-14
Medical Malpractice Seminar (Southern Medical Association), *Arlington*. Fee, $220/SMA members, $275/nonmembers. Jeanette Stone, PO Box 2446, Birmingham AL 35201, (205) 323-4400.

October 18
Antimicrobial Therapy of Life-Threatening Infections (Eastern Tennessee State University), *Bristol, Virginia*. Raymond Massengill, EdD, Bristol Memorial Hospital, 209 Memorial Dr., Bristol TN 37620, (615) 968-1821.

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Annie and Oliver: Not Just a Hit Show

Eloise C. Haun, MD, *Woodstock, Virginia*

"These children reared in abusive environments are reproducing themselves in epidemic proportions. They are the walking wounded in the prison and mental health systems, the failures in the work force, the survivors in the chronic-care facilities. The economic burden is phenomenal."

PARENTAL ASSAULT is only symptomatic of an abusive environment. The resultant psychiatric lesions are pervasive in the victim and extend to future generations, affecting our society and its history.

The abused child, Jim Jones, dramatically exemplifies the powerful malignant dynamics which magnetized the misery of similar human beings into a lethal cult. The inward-directed rage could well be directed outward, and often is directed in violence and destruction on the human race by these children, individually and collectively. These children reared in abusive environments are reproducing themselves in epidemic proportions. They are the walking wounded in the prison and mental health systems, the failures in the work force, the survivors in the chronic-care facilities. The economic burden is phenomenal.

Child psychiatry and its allied disciplines are equipped with the scientific expertise to interrupt and modify the process. Social scientists are learned on issues of cause and effect. The national health funding allocations continue to be token. The courts continue to complicate the problem. The abusive parent is assumed to have loss of impulse control due to psychosocial stressors. Culpability and responsibility for the welfare of the child are not upheld. The vulnerable child has no voice or credibility. Public assistance is often provided to the parent, ostensibly to relieve stress and make life more comfortable for the perpetrator of abuse. Is this naivete or expedience? Behavioral scientists would label this "reinforcement."

Address correspondence to Dr. Haun at 336 South Main Street, Woodstock VA 22664.

Submitted 5-12-83.

SO MUCH FOR WAILING and moaning about it. Let's look at what we know. Even without fractures and hematomas, the abused infant shows failure to thrive, psychogenic megacolon, projectile vomiting, rumination, and eczema.

Then there is the curious psychosexual dwarfism, or reversible hyposomatotropinism seen in children from abusive homes. It is manifested by failure of statural growth and mental development. Deficient growth hormone has been identified. No specific

nutritional deficiency is reported. Related findings include pain agnosia and accommodation to somatic trauma, elective mutism, eating disorders, polydipsia, sleep disorders, elimination disorders, and behavioral pathology. The intelligence may be subnormal. Socialization is retarded. Motor skills are below normal. Delayed puberty occurs. Removal of the afflicted child from the abusive environment totally reverses the syndrome. Recovery is documented within weeks. During the recovery phase, hyperkinesis and limit testing are observed followed by steady progress in statural and intellectual growth.[1]

We can cite further findings in physical examination. Even in children who have not sustained head trauma, neurological dysfunction can be diagnosed in greater than one-third of the cases of the abused. Violent juvenile delinquents from abusive homes have shown major neurologic phenomena, such as choreiform movements, positive Babinski signs, inability to skip, abnormal electroencephalograms and impressive impairment with discrepency testing in laterality.[2]

UNPREDICTABLY subjected to physical and verbal assault, the child develops psychiatric sequelae which are predictable. Coping mechanisms are generated in anticipation of danger. Mental energy is invested in hypervigilance as an adaptation to impending disaster.

These children acquire expertise at perceiving the mood of adults. Separate sessions for evaluation by different clinicians will bring about conflicting behaviors. It is pathognomonic of abused children that their behavior is not a good indicator of their inner selves. Precocious skills are evident in these children's initiation of a relationship with a strange adult. Pseudo-adult mannerisms often surface. Instant rapport, followed by the child's testing of the interviewer, is the usual experience of the psychiatrist. The normal oppositional independence of the two-year-old is ominously missing. These same two-year-olds do not show social reserve but will coyly approach strangers with friendly overtures.

When these children are tested, they often show passive resistance. Faking friendliness converts to faking ignorance and incompetence to avoid censure. During testing, the child will narrate all his moves in advance so as to test the examiner's reaction.

Exploring the abused child's thoughts and feelings through art and play can be frustrating. This modality can be useful at a later time when the child has been removed from the abusive environment and has had frequent contact with a psychotherapist to establish trust. We find the abused child to be incapable of spontaneity, creativity, free play and collaborative play. At best, a sullen, obsequious participant on task is seen. Some children will endure long testing without protest but certainly without interest or joy. Typically, an abused child will not explore a toy box or playhouse. He is distracted by his defenses, fears, and fantasies. Pursuit of the unconscious requires a larger sampling of behavior because the child is less available for comprehension.

How so? Several mechanisms are operant. Identification with parents who relate poorly to others and have no joy in relationships occurs. Second, no experience in being supported and appreciated in trying to do anything is familiar. Instead, the child is denigrated for imperfect mastery or investigation. Self-confidence and perception is poor. There is another pervasive influence. The child is never permitted to exercise autonomous ego functions. Many, if not most of these children, are indoctrinated with confusing perceptions of reality. The parent projects into the child his own unacceptable qualities and, believing this, punishes the child for the quality the parent loathes in himself. The child has no identity except what the parent allows him to have at the moment.

BEHAVIORAL PROBLEMS inevitably surface in school, foster care, or other settings. When removed from the abusive parent, these children are in alien territory. They have no trust or consistent object relations. Self-control is lost and violent aggressive outbursts occur. The external controls lifted, the repressed rage emerges.

Superego structures are deficient. These children have no sense of social code. Transgressions of their own are perceived as survival skills, or necessary behaviors in response to stress from others. Responsibility for acts of aggression is rarely acknowledged. Denial and distortion of behavior is commonplace. Rehabilitation of abused children is difficult in foster care because of the highly disturbed behavior which emerges. Residential psychiatric care often provides the buffer of expertise to absorb the pathology and treat it on a daily basis.

Psychotic phenomena are commonplace. Reality to the child is interpreted by the adult. Often the parent has repeatedly denied the child's perceptions and presented distortions. Another internal mechanism, called "vertical splitting of reality testing," occurs.

"If the very parent who abuses and is experi-

enced as bad must be turned to for relief of the distress that the parent has caused, then the child must, out of desperate need, register the parent delusionally as good. Only the mental image of a good parent can help the child deal with the terrifying intensity of fear and rage which is the effect of the tormenting experience. The alternative—the maintenance of the overwhelming stimulation and the bad parental imago—means annihilation of identity of the feeling of the self. So the bad has to be registered as good. This is a mind-splitting or a mind-fragmenting operation."[3]

Schooling is obviously difficult. Learning has been compromised and distorted. Attention to task is underdeveloped. Trusting and learning from lan adult is impaired. Inner turmoil continues to distract attention, retention, and recall. Memory is contaminated by flooding anxiety.

Depression is an inevitable component of the healing process. Loss of family, identity, and childhood is felt by the survivor-victim. There is no history to recapitulate with loving relatives. It is an empty graveyard, the retrospective muse.

SEXUAL ABUSE requires special attention because of the unique family and social ramifications. The child is forced to be compliant without recourse, to lie for secrecy. To tell is to devastate mother, destroy the family, imprison father. Often the female child is advised that because she submits, her siblings will be spared; or that father will not have to meet his needs outside the home. Furthermore, normal protest is unavailable because of incredulity. Victims of incest are clustered at a mean age of eight years old—third graders. To share the information with peers is impossible. The victim will be ridiculed by friends, if not family.

The helplessness, isolation, and rage requires adaptive mechanisms to survive. Restructure of reality to protect one's self and the parent is required. Alleviation of pain can be achieved with altered states of consciousness and dissociation. Imaginary friends can be contrived. Multiple personalities, such as the suffering helpless individual, the angry bad girl, the sexual predator, and the sweet loving girl can evolve.

When defenses break down, self-destruction, suicide, mutilation of the self, sexual promiscuity, running away and substance abuse are alternative. A patient claimed that alcohol allowed her to feel pain, and drugs allowed her to be anything she wished.[4] Exploiting father for pay and favors validates the girl as the whore she perceives herself to be.

Hatred of mother for failure to protect her causes the girl, whose role model is her mother, to despise herself as a female. Soliciting and craving male attention to obtain self-love becomes reinforced.[5]

Adult women with an incestuous childhood frequently have symptoms of hysteria, sociopathy, domestic martyrdom, altered consciousnes, and rage reactions.

Hospitalization of abused children for diagnostic assessment is indicated.[6] Beyond the identifiable physical trauma lie the incapacitating psychic wounds. Multilateral observers on a longitudinal basis may collaborate to present the court with a recommended course of therapy. Funding to implement the plans requires special attention. Therapeutic foster care clearly requires additional support for the foster parents. Residential treatment may be an initial asylum on the road to rehabilitation.

SO WHAT about Annie and Oliver? Is Daddy Warbucks a cruel joke for every hurting child's fantasy? Did Oliver discard the tutelage of Fagan when rescued from his miserable plight? What is the story behind the hit show, the entertainment of the masses?

Perhaps it is not the quagmire of mental illness and suffering. Perhaps it is that all of us find joy in hope and healing. Perhaps in all of us there is a victim of something waiting to be rescued. Perhaps in all of us there is a hero, wanting to be there when called upon.

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Identifying the Child at Risk for Repeated Abuse

Frank T. Saulsbury, MD, *Charlottesville, Virginia*

Results of a four-year study of 751 abused children treated by a hospital-based team show that victims of more than one abusive incident were younger when initially injured than those abused only once. Crucial to prevention of repeated abuse of children is identification, on the occasion of the first episode, of the child at risk for continuing harm.

MAJOR ATTENTION in the area of child abuse has focused on the reporting of suspected cases and post facto intervention. While the ultimate goal is the primary prevention of child abuse, a more immediate concern is the prevention of reinjury of previously abused children. Repeated incidence of abuse is known to occur in a substantial proportion of abused children[1-6], and prevention is possible only if children at risk for reabuse can be identified at the time of their first incident.

Multidisciplinary teams play an important role in the identification, treatment, and prevention of child abuse[7]. This study examines two groups of children reported to a hospital-based, multidisciplinary team in order to identify the variables associated with reabuse.

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Methods

The multidisciplinary Committee for Child Protection receives all reports of suspected child abuse and neglect originating within, or referred to, the University of Virginia Hospital. Information collected on each child includes, among other things, age, gender, race, type of abuse or neglect, alleged perpetrator, the status of protective services and juvenile court involvement. Physical abuse includes bruises, lacerations, burns, internal injuries and bone fractures. Sexual abuse includes rape, incest and molestation. Neglect includes physical, emotional, medical and educational neglect. An "at risk" category includes children from chaotic social environments with a high potential for abuse or neglect and in need of preventive-protective services. For simplicity, this report refers to all categories as abuse.

The records of the Committee for Child Protection from January 1, 1976, through December 31, 1980, were reviewed and two groups of children were identified: those who had been abused once, and those with two or more reports of abuse. The

two groups were then compared in order to determine whether there were factors that would serve to differentiate the two groups. To do this, demographic data surrounding the initial report in the group with reabuse were compared to similar data recorded in the group with a single incident of abuse. The results were analyzed statistically by means of the Chi square method.

Table 1. Variables in Incidence of Abuse and Reabuse in 751 Children.

	Reabuse N = 83	Single Incident N = 668	P Value
Age			
1 yr	36 (43%)	143 (21%)	<0.001
1–5 yrs	22 (27%)	199 (30%)	NS*
6–12 yrs	15 (18%)	144 (22%)	NS
13 yrs	10 (12%)	182 (27%)	<0.01
Type of Abuse			
Physical Abuse	14 (13%)	207 (24%)	<0.05
Bone Fractures	12 (12%)	42 (5%)	<0.01
Sexual Abuse	6 (6%)	87 (10%)	NS
Neglect	22 (21%)	208 (24%)	NS
Miscellaneous	14 (13%)	105 (12%)	NS
At Risk of Abuse	36 (35%)	212 (25%)	<0.05
Alleged Perpetrator			
Mother	36 (43%)	178 (27%)	<0.01
Father	10 (12%)	97 (14%)	NS
Other	37 (45%)	393 (59%)	<0.05
Disposition			
Protective Service	71 (86%)	507 (76%)	NS
Juvenile Court	8 (10%)	78 (12%)	NS
Remains with Parents	72 (87%)	517 (77%)	NS

* NS = not significant

Results

During the study period there were a total of 751 children referred to the committee because of abuse. There were 668 children with a single report of abuse and 83 children with two or more reports. Comparison of the two groups is depicted in Table 1. There were no significant differences between the groups in terms of race or gender. Patients ranged in age from one month to 16 years in both groups; the group with reabuse, however, was significantly younger at the time of their first reports, with a mean age of 3.8 ± 4.7 years (mean ± 1S.D.), compared to a mean age of 7.0 ± 5.7 years (mean ± 1S.D.) for the group with a single report ($P < 0.001$). The median ages were 1.5 years and 4.5 years respectively.

Forty-three percent of the children who had been reabused were less than one year of age at the time of the first report, whereas children less than one year comprised 21% of the group with a single report ($P < 0.001$). Conversely, children over 13 years of age comprised a significantly greater percentage of the group with a single report ($P < 0.01$).

Although physical abuse in general was more common in the group with a single report, the children who experienced reabuse suffered more severe injuries. Twelve percent of the children with subsequently reabused sustained bone fractures at the time of the initial episode of abuse, as compared to 5% of children with bone fractures in the group with a single episode of abuse ($P < 0.01$). We found no differences between the groups in terms of the percentage of reports for sexual abuse or neglect; at risk referrals, however, constituted a significantly greater proportion of reports in the children with subsequent reabuse ($P < 0.05$). In general, the type of abuse remained constant in the reabuse group. For example, children who were physically abused initially suffered subsequent physical reabuse, and sexually abused children tended to suffer repetitive episodes of sexual abuse.

The mother was designated as the alleged perpetrator in 43% of the children reabused and 27% of those abused only once ($P < 0.01$). There were no significant differences between the groups in terms of protective services and juvenile court involvement or foster care placement.

The committee received a total of 206 reports on the 83 children with multiple episodes of abuse. All had at least two reports, 23 had three reports, nine had four reports, five had five reports and three had greater than five reports during the study period. As shown in Table 2, the intervals between reports tended to be short: less than 12 months in the majority of instances and often less than six months.

Discussion

Reabuse of children is a problem of considerable magnitude. Previous studies have estimated the incidence of reabuse to be as high as 20%-60%[1-6]. The incidence of reabuse in the present study was 11% (83% of 751); however, this figure almost certainly underestimates the true incidence of the problem in the study population. Since the study was conducted by a hospital-based, multidisciplinary team, only those episodes of reabuse

Table 2. Intervals by Months between Reports of Reabuse in 83 Children.

Months	2nd Incident	3rd Incident	4th Incident	5th Incident
1–6	35 (42%)	10 (43%)	7 (78%)	3 (60%)
6–12	16 (19%)	8 (35%)	1 (11%)	2 (40%)
12–24	22 (27%)	5 (22%)	1 (11%)	0
24–36	5 (6%)	0	0	0
36–48	5 (6%)	0	0	0
	83	23	9	5

CHILD ABUSE AND NEGLECT

THE PROBLEM

Child abuse and neglect is a serious problem in our country. Over 39,000 reports of abuse or neglect were received by welfare agencies in Virginia during the twelve months ending June 30, 1982. All of the reports were investigated and approximately 8,200 cases were "founded" – a term used by welfare officials in cases where there is enough proof of abuse or neglect to justify serious protective measures on behalf of the children involved. In approximately 4,400 additional cases the children involved were found to be "at risk" – meaning that sufficient proof of abuse or neglect did not exist but circumstances in the family indicate that abuse or neglect is likely to occur.

THE LAW

Virginia law provides that: " 'Abused or neglected child' shall mean any child less than eighteen years of age whose parents or other person responsible for his care:

1) Creates or inflicts, threatens to create or inflict, or allows to be created or inflicted upon such child a physical

or mental injury by other than accidental means, or creates a substantial risk of death, disfigurement, impairment of bodily or mental functions;

2) Neglects or refuses to provide care necessary for his health; provided, however, that no child who in good faith is under treatment solely by spiritual means through prayer in accordance with the tenets and practices of a recognized church or religious demomination shall for that reason alone be considered to be an abused or neglected child;

3) Abandons such child; or

4) Commits or allows to be committed any act of sexual exploitation or any sexual act upon a child in violation of the law."

Cases of abuse and neglect may involve a number of different circumstances such as:

1) Physical abuse:
Physical harm which may be seen in bruises and cuts, welts, lesions, burns, internal injuries, broken bones and even death.

2) Emotional abuse:
Although it may be more difficult to identify than physical abuse, emotional abuse may consist of continual rejection demonstrated by ridicule or other aggressive behavior.

3) Neglect:
Child neglect can be both physical and emotional and most often is an act of omission such as (a) failure to feed or nourish a child properly, (b) failure to provide acceptable supervision, proper clothing, or suitable shelter, (c) failure to send the child to school, (d) failure to furnish adequate medical and dental care, (e) failure to provide the child with proper emotional stimulation.

4) Sexual abuse:
Perhaps the least visible area of child abuse involves the commission of sexual acts upon the child by a parent or custodian. The very nature of this form of abuse makes incidents of this nature difficult to discover and the severe physical and emotional effects of such abuse are often compounded as such activity continues over a period of months or years.

WHAT CAN BE DONE

The law *requires* certain persons to report suspected cases of abuse and neglect. Teachers, doctors, nurses, paid child care personnel, social workers, and staff members of state and private facilities where children are committed or placed for care or treatment are included in the group of professionals who must report suspected cases.

Virginia law provides that anyone may confidentially report suspected child abuse and neglect to local authorities without fear of criminal or civil liability so long as the report is not motivated by ill will or malicious intent. All reports will be investigated.

If you suspect that a child is an abused or neglected child, call your local office for child protective services or call the State "Hotline" and report what you know to a trained child protective services worker.

Counseling and other forms of assistance will be provided to families experiencing this problem. Protective measures will be taken if necessary. Children cannot protect themselves. They need our assistance.

24-HOUR STATE HOTLINE
1-800-552-7096

Presented as a public service by the Virginia Bar Association, Young Lawyers Section, the Medical Society of Virginia, and the Virginia Chapter, National Committee for the Prevention of Child Abuse.

serious enough to warrant medical intervention would come to the team's attention.

Although the issue of reabuse has received considerable attention in the past, there are large gaps in our understanding of the problem. The causes of reabuse are multiple and vary from family to family. Presumably, physical or mental handicaps in the child, serious psychopathology in the abuser, and lack of response to therapy of the abuser would put the child at risk for reabuse.

In the present study, we have identified a number of factors significantly associated with reabuse.

- Young children who suffered bone fractures, allegedly at the hands of their mothers, were more likely to suffer reabuse.
- While younger children have more opportunity to experience recurrent abuse than older children, this factor alone would not explain the findings in the present study.
- We found that the majority of injured children were reabused within 12 months of the initial episode, and many recurrences were reported within six months of the initial episode.
- The finding of serious injuries, such as bone fractures, at the hands of the mother in the children with subsequent reabuse may simply reflect increased contact of the infant with the mother. Alternatively, this finding may be symptomatic of a more seriously disturbed mother-infant relationship in this group.
- The increased percentage of children categorized as at risk in the group with subsequent reabuse is not surprising. This group is comprised of children from very chaotic social situations; many were initially reported to the committee at birth or shortly thereafter for preventive-protective services, and many were reported because of chronically abusive situations.

Despite some striking differences, the two groups were similar in other respects. We found no differences between the groups in terms of the percentage of cases with either protective services or juvenile court involvement, or the proportion of children who remained in the custody of the parents. We do not have detailed information regarding the types of rehabilitative services offered to the families in either group. Specifically, it is not known whether treatment was any different in the group with subsequent reabuse from the group with a single episode. Regardless of whether the cases were handled differently, our data show that the ultimate disposition for the children was similar in both groups.

Previous work by Morse et al[2] has shown that physical and emotional problems in the child and serious psychopathology in the abuser are factors associated with repeated abuse. To date, efforts designed to prevent reabuse and to rehabilitate the family have been disappointing, as evidenced by the high reabuse rate despite a variety of intervention strategies[1]. Factors associated with unsuccessful treatment outcome included a history of childhood maltreatment in the abusive parent, severe injuries in the abused child, and involuntary entry into a treatment program[8].

The results of the present study, coupled with information from previous studies, should enable the multidisciplinary team to identify more accurately children at risk for reabuse at the time of the initial report, and early identification would enable the multidisciplinary team to intensify or modify the therapeutic efforts aimed at the families.

The author gratefully acknowledges the assistance of Judy Smith and Virginia Johns.

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Sexual Abuse of Children

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According to the literature, only a fraction of the incidence of sexual abuse of children is reported. Most of the reported cases come to the physician's attention through such complications as sexually transmitted disease. The authors discuss these complications and other physical problems of the sexually abused child.

The sexual abuse of children seems to be emerging as a major problem in our society, although because of the secrecy surrounding it, its exact incidence is unknown. Kempe estimates that less than 10% of cases are recognized.[1] A nonrandomized survey of New England college students concluded that 19.2% of the surveyed females and 8.6% of the males had been sexually victimized in childhood.[2] Recent reports note that sexual abuse accounts for 9%-17% of all confirmed child abuse cases in the United States, or about 336,000 children per year.[2,3]

What is sexual abuse? As defined by Kempe,[1] it is "the involvement of dependent, developmentally immature children and adolescents in sexual activities that they do not fully comprehend, to which they are unable to give informed consent, or that violate social taboos or family roles."[1]

Data for 1981-1982 from the State Unit of Child Protective Services indicate that 7.5% of all child abuse cases involved sexual abuse, and in certain localities children seemed at somewhat greater risk. For example, in the Norfolk/Virginia Beach area during 1981-1982 there were 2,300 reported incidents of child abuse that could be verified or were highly suspicious of verification: of this total, 9%, or 210 cases, involved sexual abuse. In Hampton, the figure for the period is 5.5%, or 20 cases; for Fairfax, 6.8%, or 76 cases; and for Richmond, 5.6%, or 52 cases. For Roanoke, however, the figure is 1.4%, or 12 cases. The data for 1981-1982 indicate that the incidence of sexual abuse increased not only as a percentage of all child abuse in Virginia but also in number of cases.[4]

The majority of sexually abused children are prepubescent,[2,5-7] Children who have been sexually abused are more likely to be females, although males account for approximately 14% of cases. Studies indicate that the incidence in females peaks

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at 5-6 years and then again at 14-15 years, while males have a single peak at 6-7 years.[2,5,6]

Suspicion of abuse can be triggered by a host of behavioral and medical clues and often from statements of the victim. In Virginia, an estimated 13% of the confirmed cases were first discovered when victims presented with related health problems. Pregnancy accounted for 1.5% of these problems, sexually transmitted diseases for 4%. Other studies indicate that vaginal discharges or other signs of infection or irritation account for the majority of disclosures.[3,8]

In the male victim, anal problems such as fissures, constipation or condyloma acuminata are often the first clue that sexual abuse has occurred.[9] Vaginal discharge in the premenarcheal female is often the only indication. In one study, 15% of premenarcheal females presenting with a vaginal discharge had a culture-proven *Neisseria gonorrhoeae* infection, strong evidence that sexual abuse had occurred.[8]

Recent national reviews of sexually abused prepubescent children have reported that fondling of the genitalia and oro-genital contact were the predominant acts, but in Virginia, fondling and vaginal intercourse have been the predominant acts.[6,7,10]

Significant physical trauma requiring hospitalization occurs in less than 2% of all confirmed cases. Of those hospitalized, the majority of children are between 3-5 years and 12-14 years of age. However, 13% of the assaulted children aged 2 years and under require hospitalization.[2,3,5-7]

The composite of this data is a school-aged female who has been fondled or coerced into vaginal contact with a caretaker, in which no violence was used and no physical trauma was caused. If a male was assaulted, anal intercourse or oro-genital contact had occurred.[8-12]

Complications

Reports from the Centers for Disease Control suggest that although Virginia's statewide rate of reportable sexually transmitted diseases is slightly below the national average, certain areas of the state are experiencing much higher rates, and these are much the same areas that report a high incidence of sexual abuse of children. Moreover, it is estimated that for every case of gonorrhea, there is at least one case of non-specific vaginitis or urethritis.[13] It is not surprising, then, that many victims of sexual abuse also contract a sexually transmitted disease.

Estimates of the occurrence of gonorrhea in abused children vary from 2.8% to 53.8%.[14,15] Rimsza and Niggeman found gonorrhea infections in 7.4% of their 311 patients.[7] A recent study by Ingram et al found that 100% of children ages 4-12 with gonorrhea had a positive history of sexual activity, usually with an infected male member of the extended family.[16] Virginia law requires that sexual abuse be considered in all cases of prepubescent children with proven gonorrhea infections, excluding neonatal ophthalmia.

Anal and oro-pharyngeal gonorrhea are frequently asymptomatic and the only way to make the diagnosis is by having a high index of suspicion and then by culture.[17,18] Ingram found positive rectal cultures in seven of 17 girls and positive oropharyngeal cultures in two of 16 girls, none of whom gave a history of this type of sexual contact.[16]

Recently we have noted that *Chlamydia trachomatis*, *Mycoplasma hominis*, and *Ureaplasma urealyticum* infections are also being seen in abused children. These organisms are responsible for what physicians usually diagnose as non-specific vaginitis. Herpes simplex virus has also been reported in some cases.

A review of 34 cases of condyloma acuminata suggests that the causative virus, genital wart virus, can also be spread by sexual contact. In two cases, transmission was via anal intercourse, with the patients, both males, presenting with rectal bleeding.[19]

Evaluation

Guidelines have been published for the psychological, social, forensic and medical evaluation of children who are suspected of having been sexually abused.[2,15,20]

In Virginia, the Physical Evidence Recovery Kit for collection of forensic samples is available through local health departments. Proper use of the kit insures the proper handling of specimens. This is essential for the establishment of a bona fide chain of evidence for court.

Oro-pharyngeal cultures should be made by swabbing the entire oral cavity, not just the pharynx. Swabbing under the tongue and along the gum line has increased the likelihood of finding spermatozoa after oro-genital contact; swabbing the same area should also increase the finding of *N gonorrhoeae*.[2,11,12,15,20]

All of this should be accomplished prior to any mouth rinsing that is done for collection of physical evidence.

In asymptomatic children, urethral cultures can be made by swabbing the urethra with a wire loop

Table 1. Therapy for Sexually Transmitted Diseases[24–26].

Gonorrhea, uncomplicated, genital	<45kg	Amoxicillin 50mg/kg po once with probenecid 250mg/kg po once, or Penicillin G procaine 100,000 U/kg IM with probenecid 25mg/kg po once
	≥45kg	Amoxicillin 3gr po with probenecid 1.0gr po once or Penicillin G procaine 4.8 million U in two doses IM with probenecid 1.0gr po
Anal or pharyngeal		Penicillin G procaine and probenecid as per above doses
Sexually active adolescents		Amoxicillin 3g with 1.0g probenecid po once and tetracycline HCl 500mg qid for 7 days
Syphilis (+VDRL), primary, secondary or less than 1 year duration		Penicillin G benzathine 2.4 million U in two doses once or Penicillin G procaine 600,000 U IM/day for 8 days or Tetracycline HCl 500mg qid for 15 days
Nonspecific urethritis or vaginitis		Tetracycline HCl 500mg qid for 7 days or Erythromycin 10mg/kg qid for 7 days
Trichomoniasis		Metronidazole 2g po once or 250mg tid for 7 days or Clotrimazole two 100 mg vaginal tablets qhs for 7 days
Vulvo-vaginal candidiasis		Clotrimazole 100mg intravaginally qhs for 7 days or Miconazole 2% cream 5g vaginally qhs for 7 days

or calcium alginate swab. A urine sample may also be cultured. The child should be encouraged to void into a container without first washing the meatus or perineum. Only the first few drops should be collected and plated; if the collection was unsupervised, then the urine should be spun and the sediment cultured.[12]

The preceeding is not true for the premenarcheal female with a vaginal discharge, who should undergo a reasonably complete gynecologic exam and have endocervical cultures done by swabbing the outer canal with a dry swab. In addition, a Gram stain of the discharge should be examined for the characteristic gram negative intracellular diplococci.

In older females, a Gram stain of cervical secretions is less sensitive (55%-65%), and Gram stains of rectal or pharyngeal discharges are useless in making the diagnosis of *N gonorrhoeae* infection.[17,21]

Anal cultures are made by inserting a saline-moistened swab 1.5-2 cm into the anal opening, rotating it gently and holding it there for 15-20 seconds. When there are rectal complaints or symptoms, anoscopy is done to look for significant injury. Any discharge seen should be carefully swabbed and cultured.[11,17,21]

N gonorrhoeae is fragile and culturing must be done carefully. The use of Transgrow® medium is acceptable for male urethral cultures but not for oro-pharyngeal, cervical or rectal cultures. The best results from these areas are obtained by swabbing directly on Thayer Martin or chocolate agar plates, then storing these plates in 10%-15% CO_2 at 37°C.[21] In lieu of this, it is recommended that saline-moistened swabs be used for culture and sent without delay to the bacteriology lab.[22]

Patients not given antibiotics should be examined again in four days since the identification of *N gonorrhoeae* will often take 48-72 hours. Those treated with antibiotics should be recultured in 7-10 days (especially oro-pharynx and rectum) for a test of cure.

Further evaluation is by use of a 10% potassium hydroxide fungal prep and a wet (normal saline) prep. In premenarcheal females, *Candida albicans* or *Trichomonas vaginalis* have not been reported to cause vaginitis, but the wet prep might be positive for "clue cells", epitheleal cells with a stipled or granular appearance thought to be due to adherent bacteria, suggestive of *Gardnerella vaginalis*.[8]

A VDRL or other test for syphilis should be drawn at the first visit. Since *Treponema pallidum* has an incubation period of 10-90 days, it is recommended that this be repeated at monthly intervals for at least three months.[3]

Cultures for *Chlamydia trachomatis* are not widely available. If possible these cultures should be taken from urethral discharges and the cervix.

Therapy

The Centers for Disease Control recommend treatment with antibiotics based on culture results, although others suggest the use of cultures as a test of cure after antibiotic therapy.[3,15,23] If culturing techniques are reliable and the patient's family

cooperative, treatment can be based on culture results. This may be important since anal and oropharyngeal gonorrhea require more antibiotic therapy than uncomplicated genital gonorrhea.[17,21,24]

In the family who might not return, if a strong suspicion of infection exists or if cultures cannot be reliably done or interpreted, consideration should be given to the use of prophylactic antibiotics.

Which antibiotic? Since *C trachomatis*, *T vaginalis* and *C albicans* are rare pathogens in premenarcheal females, antibiotic therapy in these patients may start with procaine penicillin IM and probenecid. Tetracycline is not recommended for young children; in penicillin-allergic children, erythromycin should be used.[23,24]

Sexually active adolescents have a higher incidence of mixed *N gonorrhoeae* and *C trachomatis* infections; in this group, it is recommended to treat with amoxicillin/probenecid and tetracycline HCl to cover for gonorrhea *and* chlamydia.[23-25]

As noted, anal and pharyngeal gonorrhea are more difficult to treat. One study of pharyngeal gonorrhea in premenarcheal sexual abuse patients noted that 43% were not cured on the penicillin and probenecid regimen. If these cultures are positive, the first treatment should still be IM procaine penicillin or multiday amoxicillin.[24] If repeat cultures are positive, treatment should then be with spectinomycin. Two negative cultures one week apart after therapy verifies adequate treatment.

Procaine penicillin IM may treat an incubating syphilis infection, one recently acquired and still VDRL-negative, but a positive VDRL should be treated with penicillin G benzathine or tetracycline. Non-specific urethritis or vaginitis should be treated with tetracycline (in the pubertal child only) or erythromycin, as should any post-treatment urethritis or vaginitis.

Trichomonas vaginitis can be treated with metronidazole or clotrimazole. The latter drug or miconazole can also be used in the treatment of vulvovaginal candidiasis.[24]

The authors appreciate the help of Ann Childress of the Bureau of Protective Services.

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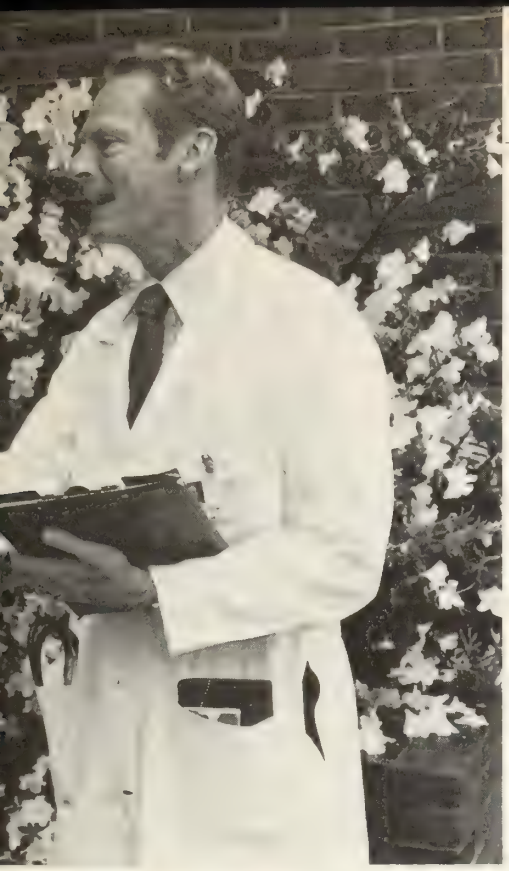
Prize Students from Virginia's three medical schools are shown here with Medical Society of Virginia Members who presented them with scholarship checks provided by the Society. Every year each school receives $2,000 from The Medical Society of Virginia for scholarships; the prize-winning students are selected by school officials.

Of the three schools, the Medical College of Virginia/Virginia Commonwealth University turned out the largest 1983 graduating class, with 161 students earning medical doctorates. Not far behind was the University of Virginia School of Medicine, where 132 were graduated. At Eastern Virginia Medical School, 95 became MDs. Women comprised more than a third of this year's graduates—100 out of 338. At all three schools, the specialities most frequently chosen for residency were internal medicine, family practice, pediatrics and surgery.

▲ At Eastern Virginia Medical School, from left to right, Charles E. Davis, Jr., MD, with student scholarship recipients Harderison Malloy, Giac-Chan Nguyen-Tan and Raymond Staniunas.

► At the Medical College of Virginia, Kinloch Nelson, MD (center), is flanked by four scholarship winners: Robert S. Miller, Evelyn Byrd Quinlivan, Amauri Gonzalez and Howard S. Ames.



▲ **At the University of Virginia School of Medicine, John A. Owen, Jr., MD (right), hands a check to Thomas Wells while a second winner, Jose Portal, watches.**

ABSTRACTS

The following abstracts were awarded prizes when they were presented at the Medical College of Virginia's Kinloch Nelson Student Honors Day on May 5, 1983.

A New Human Fibrinogen Variant with A-α Chain Defect. Joseph G. Magnant (M-85).

Congenital dysfibrinogenemia is a rare disorder in which structural and biochemical abnormalities of the fibrinogen molecule may lead to a compromised hemostatic function. We studied a 19-year-old female who bled excessively following childbirth at St. Mary's Hospital. Past and family history for bleeding tendencies or thrombosis was negative. Prothrombin time, 14 sec (control, 12 sec); activated partial thromboplastin time, 26 sec (25 sec), reptilase time, 34 sec (25 sec); thrombin time, 44 sec (19 sec). Fibrinogen was 79 mg/dl by a clotting time technique (Clauss' technique) and 384 mg/dl when measured immunologically. Similar abnormalities were found in her mother and three brothers. The kinetics of cleavage of her purified fibrinogen with thrombin, reptilase and a venom from the southern copperhead snake demonstrated markedly impaired fibrinopeptide-A release. The release of fibrinopeptide-B was normal only with thrombin. The rate of polymerization of fibrin monomers was normal. The electrophoretic mobility, as well as the immunological behavior of her reduced and non-reduced fibrinogen, were similar to the control. The amino acid sequence studies of A-α chain of her purified fibrinogen demonstrated $Arg_{16} \rightarrow$Hist substitution which appeared to be responsible for the abnormal behavior of the fibrinogen.

Production of an Anti-Thymocyte Antibody in RFM Mice with Host Versus Graft Disease by Transfer of Antibody Producing Cells from $(T_6XRFM)F_1$ Donor Spleens. Neil J. Solomon (M-85).

Host versus graft (HVG) disease is an immunologic disease produced in RFM mice by injecting newborn RFM mice with spleen cells from $(T_6XRFM)F_1$ hybrids. Host versus graft disease is manifested by glomerulonephritis, liver infarcts, enlarged lymph nodes, hepatosplenomegaly, hypergammaglobulinemia, thymic atrophy and death by day 30. Thymic atrophy with associated T-cell depletion in the spleen and lymph nodes and B-cell

hyperplasia are significant. A specific antibody has been detected in the serum of RFM mice with HVG disease that reacts against normal thymocytes. The antibody has been detected by indirect fluorescent-antibody tests. Serum from normal RFM and T_6T_6 mice are negative; but F_1 hybrids by six weeks of age have been found to produce antibody against normal thymocytes. T_6T_6 neonatal mice injected with F_1 spleen cells do not produce the antithymocyte antibody and have no symptoms pathognomonic of HVG disease. Serum from RFM mice with HVG disease reacted against thymocytes from five different strains of mice, but no reaction occurred against peripheral T-cells purified from the spleens of RFM mice using a nylon wool column to separate T-cells from B-cells. Individual serum samples from RFM mice with HVG disease had antibody titers ranging from 160 to 1280. Specific fluorescence was not changed after absorption of serum samples with liver cells, indicating the antibody lacked H-2 specificity. The antibody does not bind complement; therefore it probably opsonizes thymus cells, which are subsequently phagocytized as they attempt to seed the T-dependent regions of the spleen and lymph nodes depleted due to the allogenic reaction. Concomitantly, B-cell hyperplasia results from the loss of T-cell control as the anti-thymocyte antibody contributes to HVG disease.

Development of Methodology to Improve the Sensitivity of a Cell Culture Assay for Interferons. Catherine W. Gouldin (M-85).

The clinical significance of determining the levels of interferons in various human disorders has been substantiated. Among several criteria for selecting an interferon assay for routine use in the virus diagnostic laboratory are simplicity, rapidity, reproducibility, sensitivity, cost effectiveness, and potential for automation. Diploid fibroblasts, despite their sensitivity, do not meet all of the above criteria. This problem led us to evaluate monolayers of WISH, HEp-2, Vero, A549 and WI-26VA4 cell lines challenged with vesicular stomatitis virus (VSV) using the conventional dye uptake assay for comparative purposes. HEp-2 cells, readily available in the diagnostic laboratory, were found to be the most suitable for both IFNs, particularly HuIFN-γ. WISH cells were found to be particularly sensitive for HuIFN-α. Vero cells were the least sensitive for both HuIFN-α and HuIFN-γ.

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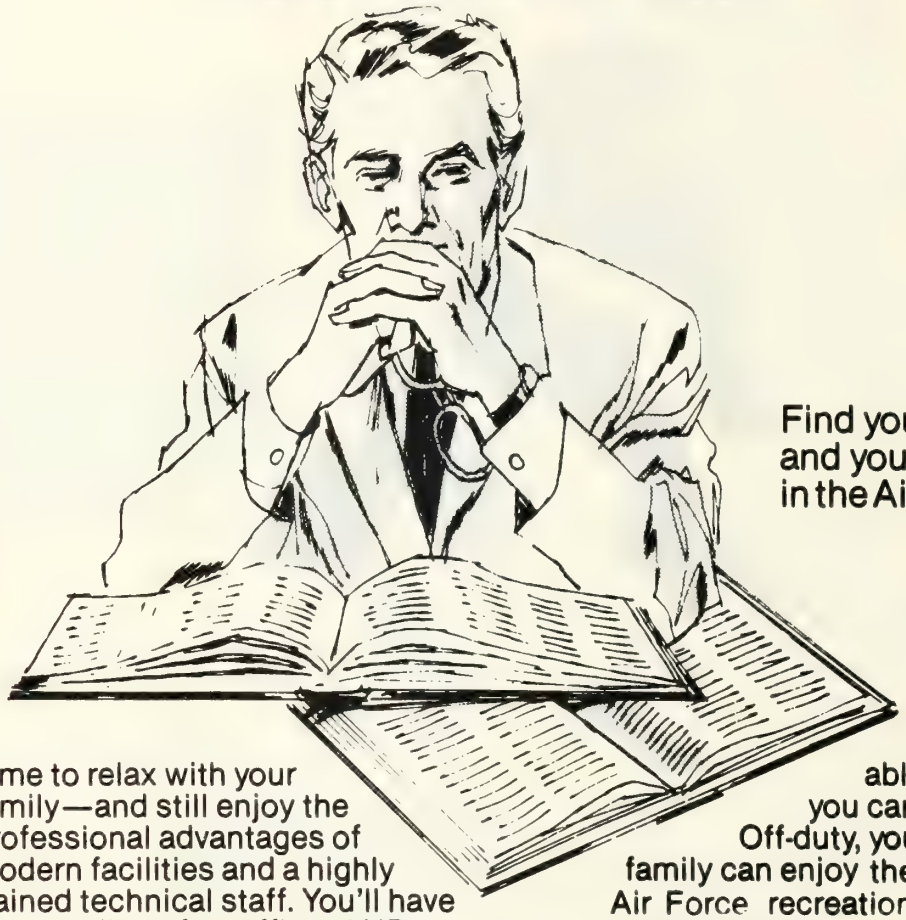

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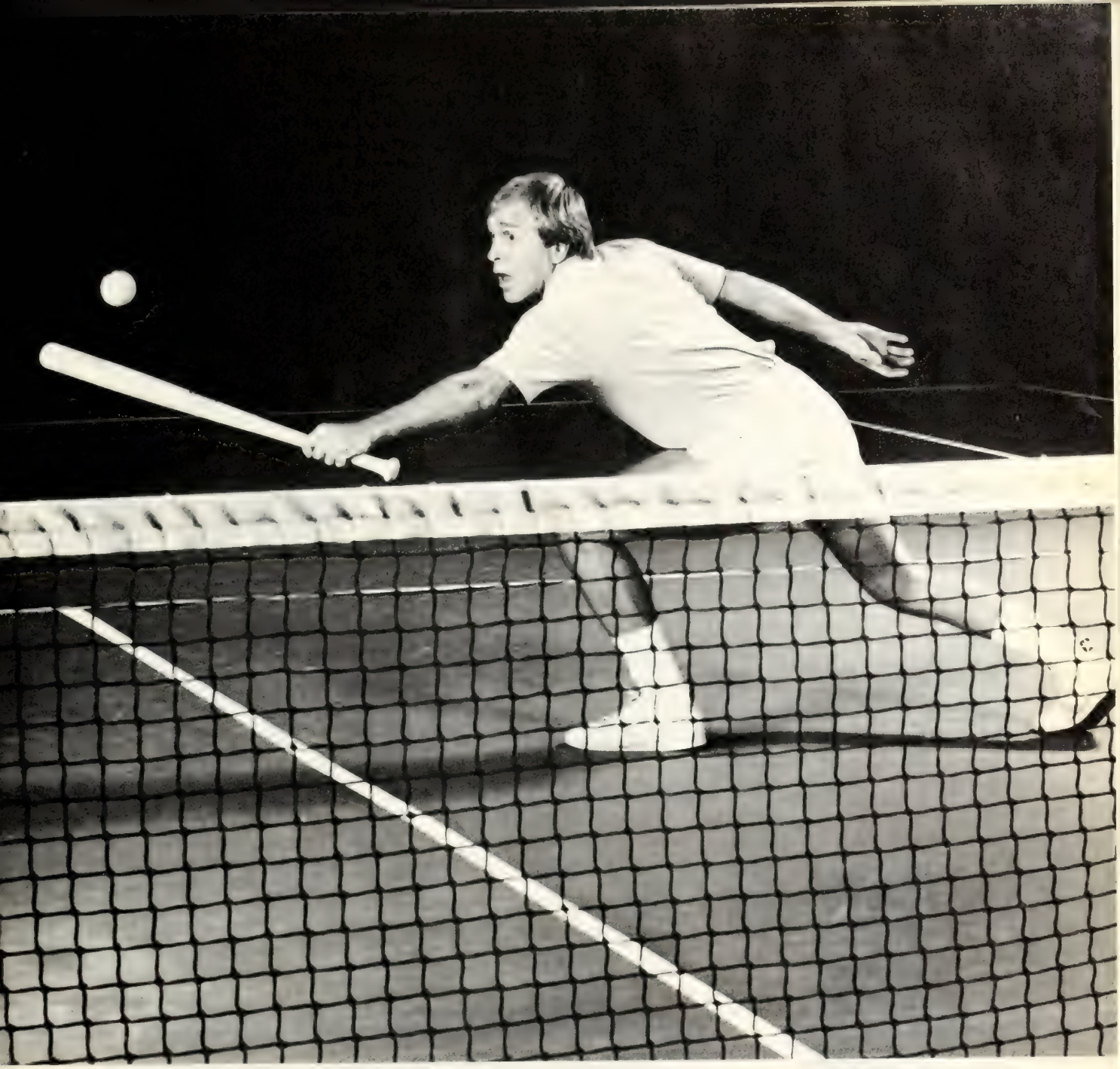

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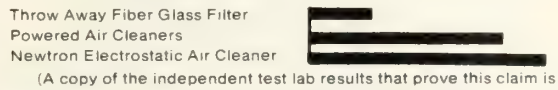
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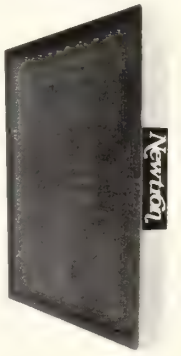

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VIRGINIA MEDICAL

EDITORIAL

Child Abuse: Aspects of a Growing Problem

In this issue there are three articles dealing with aspects of the frightening problem of child abuse: identifying the child at risk for repeated abuse, the sexual abuse of children, and the psychologic effects of child abuse. I commend them to all readers of Virginia Medical.

One specific area of child abuse that is now being recognized is sexual abuse (Ogren and Zanga in this issue). Child sexual abuse may be defined as "any sexual activity between an adult and a child. It ranges from touching and fondling to sexual intercourse." This is not an uncommon happening. It has been estimated that one out of 25 male and female children in the United States is sexually abused. Contrary to popular belief, research has estimated that 60%-75% of child sexual abuse is committed by relatives, neighbors, or acquaintances.

The American Medical Association has endorsed a program with the goals of increasing awareness of child abuse among physicians and the public: improving training and education in the use of existing methods for diagnosing, treating, and preventing abuse; promoting development of innovative programs to advance medical knowledge and skill in dealing with the problem; and encouraging physicians to work with multidisciplinary child protective teams.

At the legislative level, the Commonwealth of Virginia has exhibited great concern in regard to the sexual exploitation of children. The results of action stemming from the legislation are not yet available, but 66 multidiscipline teams are currently active in Virginia; these teams are available to help agencies work with child abuse cases and coordinate preventive services.

The "sexploitation" and sexualization of children is an additional problem. Child pornography is on the rise. A 1979 report by Densen-Gerber and Hutchinson noted that at least 264 different magazines depicting sex acts between children and adults were being sold in adult book stores across the United States. Nor does the sexualization of children stop with pornography. Children are frequently being used in advertisements in sexually suggestive poses, and movies such as "The Blue Lagoon" intimate a young teenager engaging in sexual intercourse.

The problem is an increasingly serious one and deserves the attention and concern of all of the medical profession.

Edwin L. Kendig, Jr., MD

They mobilize to fight child abuse

The brochure bound into the centerfold of this issue was developed jointly by the Young Lawyers Section of the Virginia Bar Association, The Medical Society of Virginia, and the Virginia Chapter, National Committee for the Prevention of Child Abuse.

The National Committee for the Prevention of Child Abuse was founded in 1972. It was the first organization with a nationwide focus on child abuse and neglect. There are now 32 chapters. They focus on prevention efforts, public awareness, conferences for professionals, and the development of

educational materials and a network of volunteers.

The Virginia Chapter was founded in 1980. Its board of directors is comprised of health care and social work professionals, and representatives of volunteer agencies, the business and professional community and the State Department of Social Services.

The Virginia Chapter has sponsored several significant projects. 1) Legislation was drafted to create the "Family Violence Prevention Program. The bill became law, and $800,000 was allocated to fund local programs of education and prevention.

2) Two publications will be completed this fall: a *Resource Directory* and a *Model Program Prevention Guidebook*.

3) A play entitled "Hugs and Kisses" and addressing sexual abuse will be performed by professionals around the state this fall and next spring.

The statewide effort includes the creation of 75 multidisciplinary teams of professionals and volunteers who coordinate community efforts for prevention and detection of child abuse and neglect.

Additionally, Parents Anonymous, comprised of people who have been child abusers, has nine chapters in Virginia that meet weekly under the guidance of a trained counselor.

The need to address the difficult social problem of child abuse and neglect is great. The coordinated efforts of well-motivated volunteers and professionals is an important part of the solution.

CHERYN DURRETTE

President, Virginia Chapter,
National Committee for the Prevention of Child Abuse
205 West Franklin Street, Richmond VA 23220

Reflections on Costs, Two-Tiered Care and Hospital Practice

IT IS trite but true that first-dollar coverage, Medicare and Medicaid have created a situation in this country in which noone really seems to care about the cost of medical service. I have said for years that when I was a child, if one of the nearby hospitals caught on fire, almost every patient would burn to death because they were physically unable to get out of bed and get out of the hospital, but if one of our current hospitals caught on fire, many of the patients could get out of bed and help fight the fire and would then be able to walk home. There are certainly some exceptions to this, as I found out one night when, at a banquet in Richmond, and a bright young physician across the table from me listened to my story and became quite disturbed. It turned out that he was a resident in charge of the Burn Unit at the Medical College of Virginia.

For 35 years now, young physicians entering the practice of medicine have had an easy time getting started in practice. This favorable situation has been produced by the rising expectations of patients for medical care, by the geometric rise in insurance coverage and by the governmental support of medicine through Medicare and Medicaid. Thus the cost of medical care has risen at a more rapid rate than almost any other element of our modern-day life, and we have practically achieved what I did not think possible—an end to two-tier medical care. This has been a tremendous boon to the physician, to the patient, and to the hospital, and it would be great if it could continue.

It does appear, however, that the demand for medical care is insatiable and that the dollars to pay for this care are finite. We have and are spending money that we do not have, money that we must borrow to pay for a great deal of care that is desirable but not strictly necessary from a medical point of view. The situation that has existed is that 1) the doctor has no incentive to reduce his fee, but, contrarily, the more he charges, the more he's paid; 2) the hospital has no interest in cutting costs, as it is essentially on a cost-plus payment basis; and 3) the patient has no incentive to stay out of the hospital when the insurance company is paying the bill.

There now are factors that are changing this attitude, for example, the health maintenance organizations. The HMO in Washington, DC, on the East Coast and the Kaiser Permanente program on the West Coast have long records and have been models for the dozens of new ones that have sprung up in recent years.

Independent practice associations have sprung up to compete with HMOs, but the practice patterns of the IPA's participating physicians have to change in order for this type of medical delivery system to challenge the HMOs.

The new big thing, preferred provider organiza-

tions, appears to be a step backward toward two-tier medical care but may well represent a viable and practical beginning to the solution of the rising cost of medical care.

And now come DRGs, diagnosis-related group reimbursements, discussed on pages 458-461 of this issue. The concept is intriguing, but the implementation of this principle will play havoc with cash flow and will create serious problems in the relationship between the administration and the hospital staff physicians. Administrators, of course, are much too sophisticated to appear at staff meetings and actually request that the staff reduce the number of X-rays, lab studies, special studies, etc., but there certainly will be subtle, and probably at times not too subtle, requests made of the staff to do this very thing.

Here we see a weakening of the dream of one standard of medical care for all people. It is a noble concept, and we have almost accomplished it in our country. I, for one, will be sorry to see two-tier medicine again in this country, but I do have reservations as to whether this country—or any country—can support an open-ended medical system. Socialistic countries claim to have one standard of care, but often this is a low standard for everyone, rather than the excellent care characteristic of this country. Also, most of those countries control the cost of medical care by limiting facilities—that is, they do not build hospitals, and if there are no hospital beds, there can be no patients in them. By this means they ration care and hold down costs. The certificate of need program in this country has tried to do this, but lawyers have found ways to get around certificates of need.

As the insurance companies learn that they cannot afford to sell hospital insurance with first-dollar coverage and must have substantial deductibles, and as money for Medicare diminishes and controls become more stringent, and as the DRGs begin pinching hospital income, the necessity for physician control of hospital practice will become more and more acute. The state and federal agencies with which staffs will have to deal have the force of government behind them. The hospital administrators, usually working for large corporations, have capital and legal counsels. It behooves the physicians of the hospital medical staff to join together, so that they can face the world and, specifically, the hospital leadership, in a unified and effective manner.

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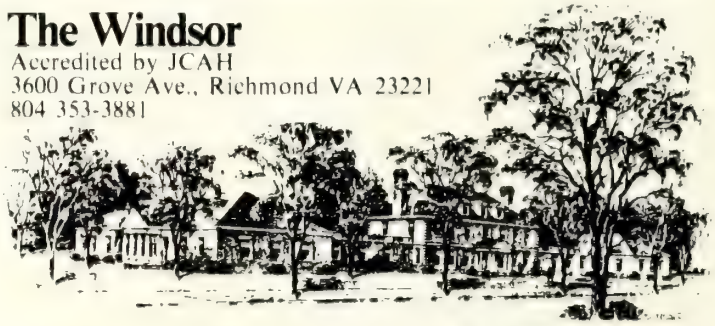

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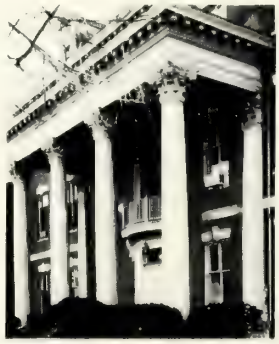

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VIRGINIA MEDICAL OBITUARY

Leroy D. Soper, MD

Dr. Leroy Dilmore Soper, former medical director of the Virginia Department of Health in Halifax and Pittsylvania Counties, died May 30 at Louise-Obici Memorial Hospital in Suffolk. He was 91 years old and had retired in Suffolk in 1965.

A native of Catlin, New York, Dr. Soper was graduated in 1915 from the University of Vermont College of Medicine, Burlington. He joined the US Army and served as a surgeon in World War I and in World War II commanded the Allied Hospital in Marseilles, France. He retired from the Army a colonel and located in Philadelphia for 15 months before moving to the Danville area in 1952 to begin his 11-year term with the Department of Health. He moved to Suffolk in 1963 and retired from practice two years later but continued to work for the Veterans Administration Hospital in Hampton and for the Red Cross.

For more than 30 years a member of The Medical Society of Virginia, Dr. Soper was also a member of the Halifax County Medical Society and the American College of Surgeons.

A. S. Thompson, Jr., MD

Dr. Albert Sidney Thompson, Jr., Richmond pediatrician, died June 17 in an automobile accident in Giles County while en route to his parents' home in Tazewell, Virginia. He was 50 years old.

A Tazewell native, Dr. Thompson was graduated from the Medical College of Virginia School of Pharmacy in 1954, worked for the National Institutes of Health in Bethesda, Maryland, and returned to the Medical College of Virginia to earn his medical degree in 1960. Following training at Richmond Memorial Hospital and the Medical College of Virginia Hospitals, he began his practice in Richmond in 1964.

He served a term as president of the Richmond Pediatric Society from 1981 to 1982. Under the auspices of the Richmond Academy of Medicine he was instrumental in developing the manual "Pre-Hospital Treatment Protocols," currently in use by Richmond area emergency medical technicians.

A member of The Medical Society of Virginia, Dr. Thompson's memberships in professional organizations included the American Medical Association, American Academy of Pediatrics and the Richmond Academy of Medicine.

John G. Maier, MD

Dr. John Gail Maier, former chairman of the Department of Radiation at Fairfax Hospital and radiologist with the Army Medical Corps for 20 years, died at age 55 on June 3 at Fairfax Hospital. He had retired last year due to ill health.

Dr. Maier, who lived in Potomac, Maryland, was born in St. Louis, Missouri, and was graduated from the St. Louis School of Medicine in 1952. That year he went to Washington DC where he joined the Army Medical Corps and practiced at Walter Reed Army Medical Center. He completed a tour of duty as commander of the 91st Evacuation Hospital in Vietnam in 1966 and 1967 and retired from the Army as a colonel in 1972. Dr. Maier then served as a clinical professor of radiology at Georgetown University Hospital until 1977, and as chairman of the Department of Radiation at Fairfax Hospital until he retired in 1982.

Dr. Maier came to membership in The Medical Society of Virginia through the Fairfax Medical Society and was also a member of the American College of Radiology.

Harold L. Goldman, MD

Dr. Harold L. Goldman, Richmond family physician, died June 3 at the age of 60.

A native of Norfolk, Dr. Goldman was graduated from the University of Virginia and its School of Medicine. He returned to Norfolk to train at DePaul Hospital and establish a practice in 1962, and 20 years later moved to Richmond. After two years as medical supervisor for Du Pont, he resumed private practice and was chairman of the family practice department of St. Mary's Hospital from 1981 to 1982. At his death he was chairman of St. Mary's pharmacy department and an associate clinical professor at the Medical College of Virginia.

Dr. Goldman was a member of The Medical

Society of Virginia, the Richmond Academy of Medicine and the Industrial Medical Association.

William R. Tyson, Sr., MD

Dr. William Roberts Tyson, Sr., longtime Norfolk physician, died April 9. He was 78 years old.

A native of Norfolk, Dr. Tyson was graduated from the College of William and Mary and the Medical College of Virginia, Class of 1935. He served with the US Army in New Guinea during World War II, returning to Norfolk to establish a practice of obstetrics and gynecology. Following retirement from private practice in 1963, he assumed a post at Hampton's Veterans Administration Hospital, remaining there until 1975. From 1975 to 1980 he worked for the Red Cross.

Dr. Tyson was a member of the Norfolk Academy of Medicine and The Medical Society of Virginia for nearly 46 years.

Memoir of William C. Gill, Jr. 1923–1982

By Lacey M. Jacobs, Jr., MD, Percy Wootton, MD, and Custis L. Coleman, MD

The medical community of Richmond was shocked and saddened on learning of the death of one of its most distinguished and beloved members, Dr. William Carlyle Gill, Jr., on July 9, 1982. Dr. Gill suffered a fatal heart attack in a Wyoming airport while returning to Richmond after visiting one of his sons.

A native of Virginia, Dr. Gill was born in Richmond and educated in the public schools. On an academic scholarship, he attended the College of William and Mary. In 1943, Dr. Gill joined the Navy, serving in the South Pacific Theater as a pharmacist's mate with the Sixth Marine Division. He was awarded the Purple Heart on Guam. After discharge from the service, Dr. Gill resumed his education at the University of Richmond, then entered the Medical College of Virginia, graduating in 1952. After serving a rotating internship, he established a family practice in North Richmond.

His contributions to the medical community are numerous. His demonstrated leadership carried him to such positions as president and board chairman of the Richmond Academy of Medicine. He chaired and served on many committees, including Budget and Mediation. In addition to his busy and highly successful private practice, his interest in academic medicine involved helping to develop the Department of Family Practice at the Medical College of Virginia, which at present is its largest budgeted department. He served for ten years as an assistant professor of family practice and as president and board chairman of the Richmond Academy of Family Planning.

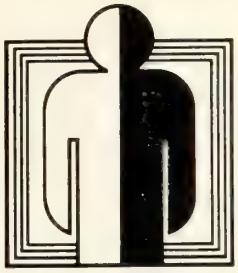
He served on numerous committees of The Medical Society of Virginia and was a board member of the South Central PSRO, the Virginia Mental Health Association, and Richmond Memorial Hospital, where, at the time of his death, he was chief of medical staff.

He married Mildred Anderson of Catonsville, Maryland, and although Bill led a very active and busy life and had many responsibilities, including being a Vestryman of St. Thomas Episcopal Church, he always arranged time for his wife and three sons. They, as well as his many friends, will miss his company, loving guidance, and attention.

Billy loved the sea and boating, not as a plaything but as something to be trusted and respected. He believed there was always something to be learned about the sea and the mystery of its depth. He served all the flag offices and was commodore of the Virginia Yacht Club. He was past flotilla commander of Flotilla 31 USCGA, which at the time was the largest flotilla in the Coast Guard, and at the time of his death he held the highest training office a member can hold. He was a member of the Boating Advisory Commission for the Governor of Virginia and taught courses ranging from basic seamanship to advanced piloting.

To have had the privilege and pleasure of knowing Billy was truly a rare and unusual experience. Memories of him will linger in the minds of those who knew him.

Prepared and published at the request of the Richmond Academy of Medicine.



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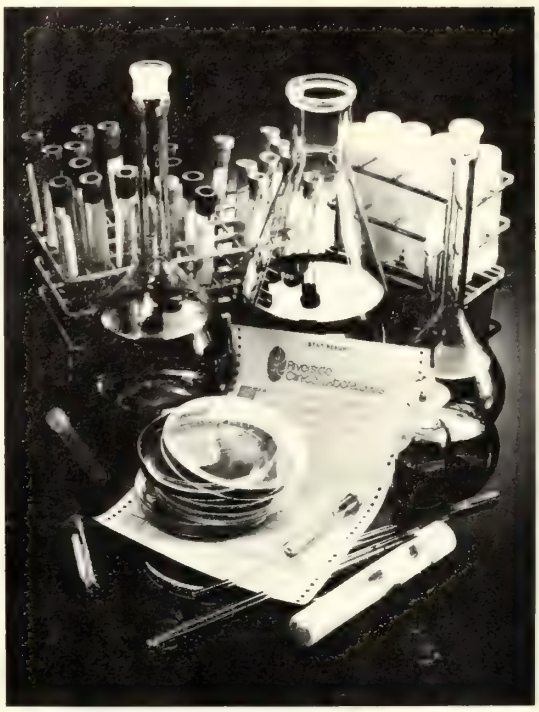

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WHO'S WHO

A classic clinician during his earning hours, **Dr. John I. Youel, Jr.**, of Charlottesville makes unconventional use of his leisure time. 1) He plays the bagpipes. 2) He goes helicopter skiing. These two unusual pursuits, plus the vascular surgeon's storied background, provided a lively feature article recently in the Richmond Times-Dispatch.

The bagpipery is a proud expression of Dr. Youel's Scottish heritage. His great-great-grandfather, William Youel, came to Virginia from Scotland in 1769. The Lebanon Presbyterian Church, which he founded, is still standing on state Route 42 near Lexington. And when his house near Goshen Pass was to be designated an historic site, with the Youel family in attendance, Dr. John Youel decided to learn to play the bagpipes for the occasion. He found his teacher in Ralph Allen, University of Virginia chemistry professor. Previous experience with the clarinet made him an apt pupil, and he progressed quickly from the introductory course with the single-reed practice chanter to the four-reed pipes.

He's been working ever since to improve his skill. Each summer he attends a bagpiping school in North Carolina, and then, turned out in Buchanan tartan kilt, he competes in bagpiping contests in the North Carolina mountains, where annual Scottish festivals attract thousands of participants.

As for the helicopter skiing, "It gets you to a mountain where you couldn't otherwise ski," the newspaper story quotes Dr. Youel as saying of the sport that uses helicopters to hoist skiers to areas where the slopes are untrammeled by chairlifts and trails.

Born in Westchester County, New York, Dr. Youel spent boyhood summers in Virginia on a farm his father bought in Albemarle County. After obtaining his medical degree at the University of Michigan and completing his training in New York, he came to Virginia to practice and built a home on his father's farm, where he now breeds horses.

Dr. James M. Wade, Abingdon otolaryngologist, is the new chief of staff of Johnston Memorial Hospital in Abingdon.

After 50 years of service at Norfolk General Hospital, **Dr. Richard B. Nicholls** is discontinuing gynecological surgery but will still be on the staff as consultant and will continue his office practice full steam ahead. Dr. Nicholls is a 1930 graduate of Jefferson Medical College, Philadelphia.

New president of the Virginia Beach Medical Society is **Dr. Bertram L. Warren, Jr.** Serving with him as president elect is **Dr. Richard A. Mladick; Dr. Thomas M. Krop** as secretary; and **Dr. Peter B. Blanchard** as treasurer.

Piper Youel in full Highland fig. *Richmond Times-Dispath photo*

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References: 1. Kales A *et al: J Clin Pharmacol 17*:207-213, Apr 1977 and data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Kales A: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **3.** Zimmerman AM: *Curr Ther Res 13*:18-22, Jan 1971. **4.** Kales A *et al: JAMA 241*:1692-1695, Apr 20, 1979. **5.** Kales A, Scharf MB, Kales JD: *Science 201*:1039-1041, Sep 15, 1978. **6.** Kales A *et al: Clin Pharmacol Ther 19*:576-583, May 1976. **7.** Kales A, Kales JD: *Pharmacol Physicians 4*:1-6, Sep 1970. **8.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **9.** Dement WC *et al: Behav Med 5*:25-31, Oct 1978. **10.** Vogel GW: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **11.** Karacan I, Williams RL, Smith JR: The sleep laboratory in the investigation of sleep and sleep disturbances. Scientific exhibit at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971. **12.** Pollak CP, McGregor PA, Weitzman ED: The effects of flurazepam on daytime sleep after acute sleep-wake cycle reversal. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, June 30-July 4, 1975. **13.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ.

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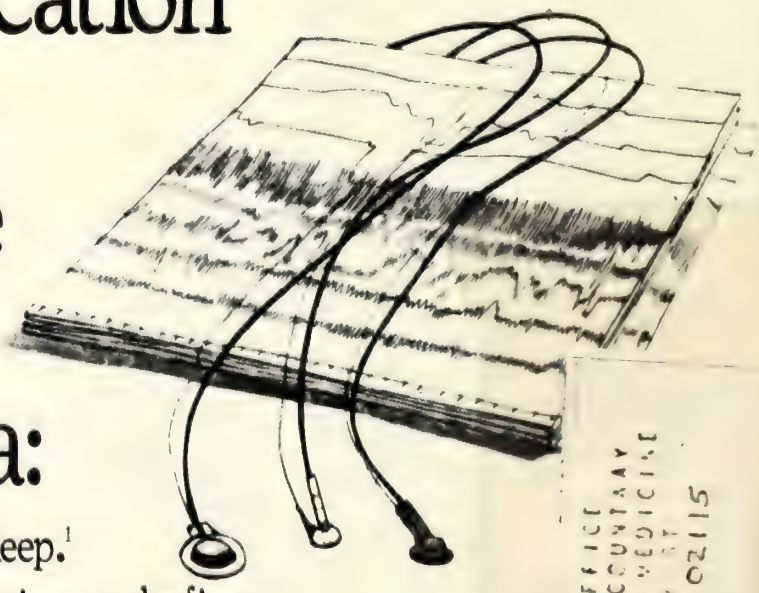

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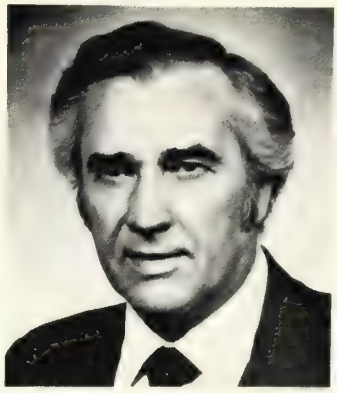

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MSV leadership coming to peer review

By Carl L. Shires

UNDER MANDATE from The Medical Society of Virginia's House of Delegates, physicians and administrators from all areas of the state are putting together a blanketing peer review plan for Virginia's doctors.

Just when these efforts will culminate in the proposed Medical Society of Virginia Review Organization (MSVRO) is not certain, for the federal government is involved. Final rules and regulations still have not issued from that source, but planning meetings are ongoing and budgetary needs have been assessed.

Tentatively, there may be some version of the plan ready for submission to the MSV Council prior to the Society's annual meeting at the Homestead in Hot Springs in November.

Cyane Lowden

At a meeting to consider the planning and development of the new Medical Society of Virginia Review Organization: left to right, Dr. Cecil F. Evans, Dr. Robert A. Morton, Dr. William M. Eagles, Dr. Robert L. A. Keeley, and Dr. John L. Guerrant.

What is taking place, in effect, is that a higher body is being readied to take over statewide direction of the peer review operation now assigned to five regional professional standard review organizations that began reviewing in 1977.

And when it all is over, there still won't be a great deal of substantive change. The existing PSROs will be the subcontractors for work, and the PSROs' physicians will continue doing the reviews, checking Medicare patient charts to determine if there are any aberrations in necessity of admission or procedure and monitoring quality of care.

If they find one of their peers isn't measuring up to local standards and practices, then there will be the usual "education." If education doesn't work, the ultimate action of sanction—a quasi-judicial proceeding replete with lawyers and due process—might ultimately come to pass. Sanctions are rare birds in the Virginia medical aviary.

The decision by the House of Delegates to go the blanket state review road is not unique. Several other medical societies are on the same route, because the Department of Health and Human Services and the Congress have indicated they would have it that way.

Again, tentatively—and most every facet of the plan still is tentative—the MSVRO's initial annual budget would be about $343,000, although that might be reduced after first-time expenses are taken care of. The money would come by taking $1.43 from each of the estimated 240,000 Medicare patient discharges in Virginia annually. The federal government

now permits $13.80 from each such discharge for the use of the regional PSROs. Still undetermined is whether regional budgets will be reduced to make way for the state outlay.

The tentative state budget would include expenditures of about $232,000 in salary and benefits for a seven-member staff; $15,000 for travel; about $25,000 in legal and accounting fees; about $17,000 for furniture, equipment and computer rental; $52,000 for support costs (rental, printing, postage, etc.); and a $50 hourly rate for meetings of board members and committees.

Recommended at a late July meeting of a Subcommittee on Planning and Development for MSVRO at Society headquarters in Richmond was the hiring of an executive director and a medical director for the new peer review organization, but subcommittee discussion revealed conflicting opinions on the necessity of a medical director at that level. That, too, ultimately will be decided.

The following physicians were present at the meeting of the subcommittee: Robert L. A. Keeley, Roanoke, chairman; Robert A. Morton, Norfolk; Cecil F. Evans; Newport News; William M. Eagles, Richmond; John L. Guerrant, Charlottesville; and C. Barrie Cook, Fairfax, MSV President Elect. Present also were four administrators from regional PSROs.

Meanwhile, another subcommittee has been at work developing articles of corporation, bylaws and organizational structure to include permanent committees. That group is addressing also the matter of tax-exempt status for MSVRO. This subcommittee is composed of Drs. Philip T. Rodilosso of Arlington, chairman, Harry C. Kuykendall of Alexandria and Joseph M. Deignan of Winchester.

In addition to those at the July meeting and on the bylaws subcommittee, these are the other physicians on the MSVRO board: MSV President Harold L. Williams, Newport News; H. C. Alexander III, Roanoke; Owen W. Brodie, Richmond; James E. Nevin, Danville; Ira J. Green, Alexandria; George R. Smith, Jr., Shawsville; and Frederick K. McCune, Virginia Beach. The board will grow to 17 members when a representative from the 4th district is chosen. Dr. Morton is temporary chairman.

It still is uncertain what, if any, economies might be effected by forming this blanket review group and subcontracting to regional PSROs, which have been costing the feds $3.5 million annually. The agenda drawn up for the June 15 organizational meeting stated the case for permitting business as usual, more or less, through subcontracts with the regional groups, which generally are governed by 15-member boards. The agenda noted that a state organization could be more "responsive to federal and MSV concerns" and that "performance responsibilities would be more clean-cut." The agenda paper said further:

". . . local physicians have for the past seven or eight years organized

An Invitation From the Chairman

The Program Committee for The Medical Society of Virginia's 1983 Annual Meeting at the Homestead cordially invites you to attend the scientific sessions on Friday and Saturday, November 4 and 5.

News in nuclear medicine and a discussion of osteoporosis will lead off Friday's session, followed by papers on lupus erythematosis and acute diarrheal illness.

The second Friday section will focus on computers in medicine, including an analysis of the practicality of medical data bases and a demonstration of the AMA Telenet system.

After starting with "What's new in antibiotics?", Saturday's session will switch to a trio of talks on PPOs and DRGs; to elucidate on the latter, F. Lynn May will travel to the Homestead from his office in the Department of Health and Human Services, Washington.

The committee has worked long and hard on the program and is confident you will find its diversity interesting and rewarding.

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AIDS ruled reportable

As the result of a regulation signed by Gov. Charles S. Robb in late July, acquired immune deficiency syndrome, or AIDS, has been placed on the list of diseases that must be reported to the Virginia Department of Health. Since last summer, Virginia physicians have voluntarily reported 18 apparent cases of AIDS, including five deaths. The list of diseases specified as reportable by the Virginia State Board of Health was published on page 349 of the June 1983 issue of VIRGINIA MEDICAL.

themselves with their own board and committee structures which are already in place and functional. Four of the five are staffed and can start performing under a subcontract instantly. The Southwest Virginia physician organization still exists though it has no staff, but with funding via a subcommittee, it probably can be functional within 90 to 120 days.

"Problems with quality of care and utilization of facilities are already known and being dealt with by these physician groups. The current PSROs have already developed adequate checks and balances between review personnel, committees and boards, so that physicians or hospitals which appear to have problems can be dealt with fairly . . . though the road has not always been smooth, these relationships have improved over time . . ."

There are other reasons for establishing a potentially stronger state review organization. According to the new DRG law, any hospital that has not contracted for peer review by October, 1984, will not longer be eligible for Medicare payment.

Additionally, physician organizations, such as the MSVRO, have been given a one-year priority consideration for PRO contracting. That means area health care facilities and insurance firms may not bid for contracts until after October, next year. The American Medical Association has urged state medical societies to vie for the contracts—in support of a policy advocating local, physician-directed, peer review.

Emanating from the July meeting were observations dealing with the DRG prospective payment system coming in October.[1] Under that system, certain hospital facilities, including rehabilitation and long-term psychiatric care units, are exempt from DRG payment strictures.

Dr. Morton had this to say on that subject: "You can be certain that hospitals will be setting up rehabilitation centers; we may be seeing all kinds of things in rehab units." Barbara Jennings, executive director of the Colonial Virginia Foundation, echoed: "Every hospital in Eastern Virginia is now putting together a rehab unit."

Said Dr. Keeley of DRGs: "It is a concept that is not valid. The best thing we can do is try to live with this until something else comes along." He noted further that "a lot of physicians are upset about taking 10,000 diagnoses and compressing them into 467" (the number arbitrarily assigned by the Department of HHS).

Of the DRG emphasis on reducing hospital lengths of stay, with the concomitant reduction in income, Dr. Keeley said this: "We're on the edge of a revolution in hospitals; there are going to be some death rattles. DRGs are the only thing that has come down the pike to draw their attention. We're going to lose hospital beds in Virginia."

In another telephone interview, Dr. Morton said the average hourly fee of about $50 paid to physicians

engaged in peer review "actually is more of an honorarium, far less than their usual hourly rate. Despite that, though, I'm amazed at the willingness with which they perform these often unpleasant duties. It generally means travel and review of medical records by a three-doctor team"—usually specialists in the same field as the physician whose charts are in review. "Hospitals," he added, "are almost always cooperative."

He, like others of his colleagues, is not yet convinced that the MSVRO needs a medical director. "If it is being done well on the local level, maybe we don't need to fill that position," he said. The tentative budget calls for $35,000 annually for the part-time position.

Peer reviews are much more apt to lead to "education" than to sanction, Dr. Eagles observed, and Drs. Morton and Keeley agreed with him. In his nearly seven years of association with the South Central PRO, Dr. Eagles said, he could recall only one instance in which discipline reached the sanction stage, and then it was for gross, flagrant disregard for patient safety. Dr. Keeley said sanctions "come only once in a blue moon." Dr. Morton talked of lawyer involvement when discipline reaches that stage, of "due process," and of the historic reluctance of physicians to deal with lawyers.

All three agreed generally that the system in existance now is a fair and an effective one. In response to a question posed to them separately, each said that they felt that Virginia physicians are "tough" enough on their physician colleagues and that quality care is the norm rather than the exception.

And Dr. Eagles had this to say concerning another DRG emphasis, the one on desired reduction of ancilliary procedures—lab work, X-rays, etc.: "I don't think that's going to make a great deal of difference. If the hospital comes around to me and says, 'Bill, you didn't need that test,' I'm going to say, 'Go to hell!' And I suspect a lot of physicians are going to say the same thing."

Conclusion: Virginia doctors might make some strategic withdrawals from the onslaught of the feds. But retreat? Never!

1. Shires CL: The DRGs are coming, the DRGs are coming. Va Med 110:458–459, 461, 1983

Hand to Holland

Virginia's Gov. Charles S. Robb will lend his political clout this month to the Democratic candidacy of Dr. Clarence A. Holland, Virginia Beach general practitioner seeking a seat in the Virginia State Senate.

Additionally, four area physicians—Drs. John J. Krueger, W. Andrew Dickinson, Frederick K. McCune and Guillermo E. Espejo—have formed a physicians' "support" unit to boost the Holland candidacy. A Holland endorsement by the Virginia Beach Medical Society was anticipated.

These and other factors, Dr. Holland told VIRGINIA MEDICAL by telephone, lead him to a "feeling of optimism" that he will prevail in the contest with both Republican and independent candidates. At stake is the Senate seat created during the redistricting in 1981.

If elected, the 54-year-old candidate would be the only Medical Society of Virginia member seated in the 140-member legislature. A current physician-senator, Dr. John W. Buchanan of Wise County, is not an MSV member.

In traveling to Virginia Beach this month for a "fund raiser", Governor Robb is returning political favor. Dr. Holland, Virginia Beach political insider and former mayor of the resort city, headed Robb's gubernatorial campaign in that city.

VIRGINIA MEDICAL

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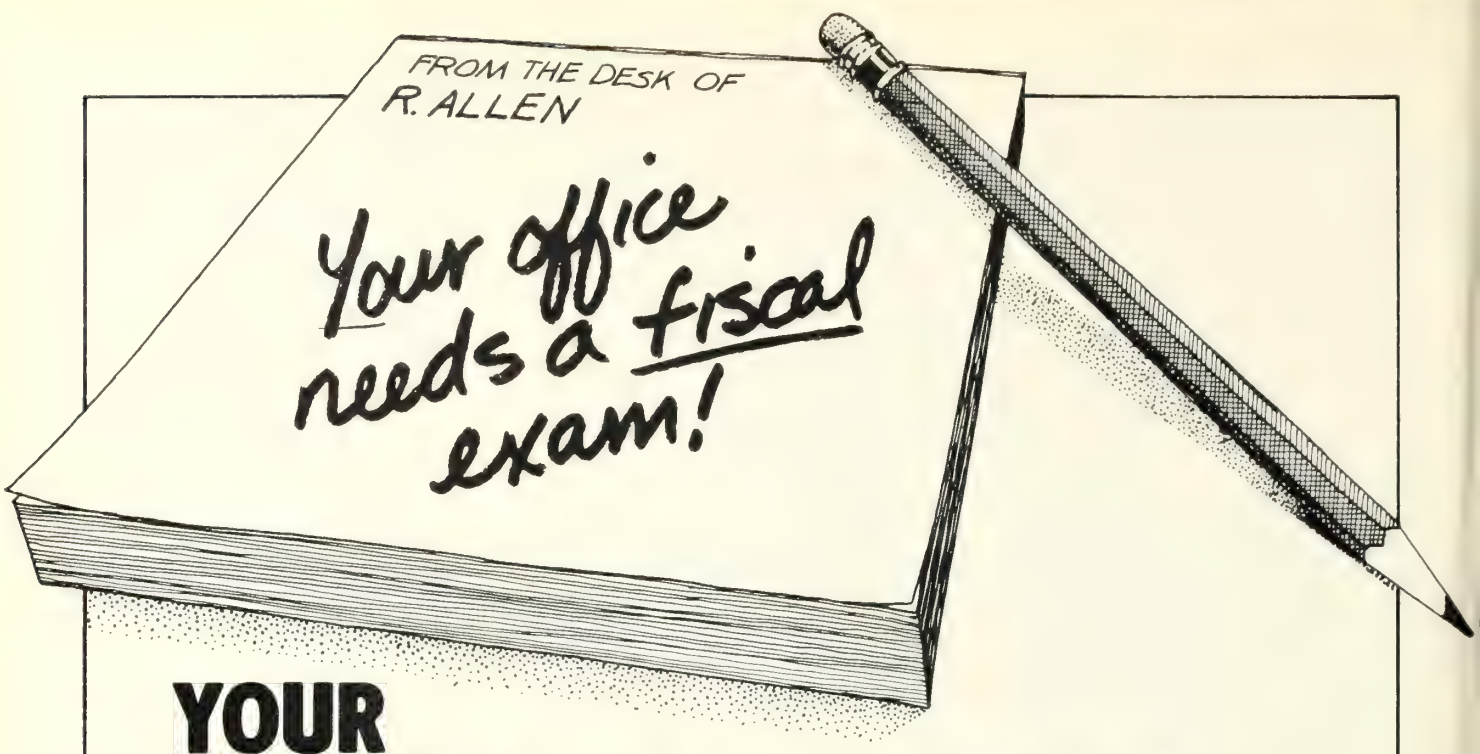

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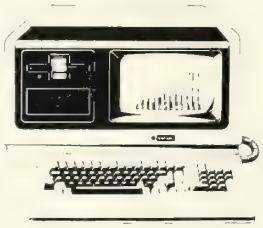

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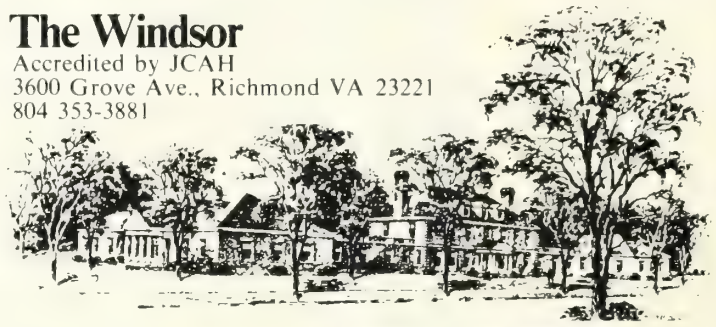

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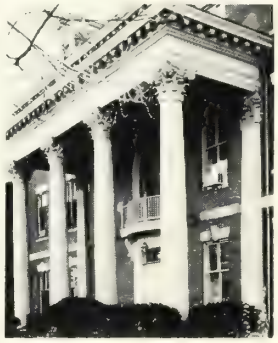

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MEETINGS ABOUT MEDICINE

September 25-28
Radiology Update (University of Virginia), *Charlottesville*. 20 credit hrs. Fee: $350. T. E. Keats, MD, Box 170, UVa Medical Center, Charlottesville VA 22908, (804) 924-2781.

September 26-30
Basic Practices for the Novice Ultrasonographer (Johns Hopkins University), *Baltimore*. 40 credit hrs. Fee: $400. Office of CME, 720 Rutland Ave., Baltimore MD 21201, (301) 955-8450.

September 27-29
Treatment of Hyperglyceridemia (National Institutes of Health), *Bethesda, Maryland*. Mrs. Peter Murphy, 2115 E. Jefferson St., Suite 401, Rockville MD 20852, (301) 468-6555.

September 30
Cardio-Pulmonary Diseases: Recent Advances (University of Maryland), *Baltimore*. Michael E. Wessely, Program of CME, 10 S. Pine St., Baltimore MD, 21201, (301) 528-3956.

September 30-October 1
Update in Rheumatology (Arthritis Foundation, Maryland Chapter), *Baltimore*. 9 hrs. Cat. I. Karen Goodman, 12 West 25th St., Baltimore MD 21218, (301) 366-0923.

October 6-8
Topics in Gastroenterology and Liver Disease (Johns Hopkins University), *Baltimore*. 21 hrs. Cat. I. Fee, $295. Noreen Javornik, Office of CME 720 Rutland Ave., Baltimore MD, 21205, (301) 955-6046.

October 7
Selected Topics in Pediatrics (Eastern Virginia Medical School/Children's Hospital of the King's Daughters), *Norfolk*. Cat. I credits. Jean E. Shelton, MD, 800 W. Olney Rd., Norfolk VA 23507, (804) 628-3798.

October 7-9
Symposium on Sarcoidosis (University of North Carolina), *Asheville, North Carolina*. 16 Cat. I credits. Fee: $275. Office of CME, 231 MacNider Bldg., UNC School of Medicine, Chapel Hill NC 27514, (919) 962-2118.

October 10-14
25th Annual Emil Novak Memorial Course in Clinical Gynecological Pathology, Obstetrics and Genetics (Johns Hopkins University), *Baltimore*. Carlita Kearney, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

October 12-14
Topics in Ambulatory Medicine (Johns Hopkins University/Baltimore City Hospital), *Baltimore*. 20 hrs. Cat. I. Fee, $275/MDs, $150/residents. Susan Bavaro, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-6046.

October 12-15
8th Annual Symposium on Advances in Cancer Treatment Research. (University of Maryland), *Baltimore*. Program of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

October 13-15
Child Neurology Society, Annual Meeting, *Williamsburg*. National Office, Box 486, Minneapolis MN 55455.

October 13-14
Medical Malpractice Seminar (Southern Medical Association), *Arlington*. Fee, $220/SMA members, $275/nonmembers. Jeanette Stone, PO Box 2446, Birmingham AL 35201, (205) 323-4400.

October 18
Antimicrobial Therapy of Life-Threatening Infections (Eastern Tennessee State University), *Bristol, Virginia*. Raymond Massengill, EdD, Bristol Memorial Hospital, 209 Memorial Dr., Bristol TN 37620, (615) 968-1821.

October 31-November 2
Mechanisms of Neurotoxicity (Johns Hopkins University), *Baltimore*. Jacqueline Corn, Continuing Education, 615 N. Wolfe St., Baltimore MD 21205, (301) 955-2609.

November 2
Management of Stress (East Tennessee State University), *Bristol, Tennessee*. Carol Blevins, Bristol Family Practice Clinic, 100 Arapahoe Dr., Bristol TN 37620, (615) 968-5212.

continued over

November 2-4
Advanced Obstetrics and Gynecology (Johns Hopkins University), *Baltimore*. 24 Cat. I credits. Fee: $300. Joan Batt, Dept. of Radiology, Johns Hopkins Hospital, Baltimore MD 21205, (301) 955-8450.

November 3-5
Antenatal Diagnosis and Treatment of Fetal Disease (Medical College of Virginia), *Williamsburg*. 14½ Cat. I credits. Fee: $275 for MDs; $175 for residents, interns. Sheri Rosner, Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

November 4-5
Asthma (American Lung Association of Virginia, Southwest Region/East Tennessee State University), Abingdon. Anne Hall, Virginia Lung Association, 524 Cumberland, Bristol VA 24201, (703) 669-2933.

November 5-6
Reading Retreat: Recent Advances in Cardiology and Gastroenterology (Virginia Chapter of American College of Physicians), *Roanoke*. 15 Cat. I credits. Fee: $100. Daniel N. Mohler, MD, University of Virginia Medical Center, Box 395, Charlottesville VA 22908, (804) 924-5118.

November 6-9
Annual Meeting: American Physicians Art Association, *Baltimore*. S. W. Parks. MD, 710 Myrtle Rd., Charleston WV 25314.

November 6-9
77th Annual Scientific Assembly: Southern Medical Association, *Baltimore*. Ms. Jeanette Stone, SMA, PO Box 2446, Birmingham AL, 35201, (205) 323-4400.

November 9
Respiratory Care Update (American Lung Association of Virginia, Richmond Region/Virginia Commonwealth University School of Nursing), *Richmond*. Shirley Lipscomb, American Lung Assoc. of Virginia, PO Box 7065, Richmond VA 23221, (804) 355-3295.

November 11-13
Update in Infectious Diseases (Johns Hopkins University), *Ocean City, Maryland*. 10 Cat. I credits. Sandy Young, Office of CME, 720 Rutland Ave., Baltimore MD, 21205, (301) 955-5880.

November 15-20
Gynecologic Endoscopy and Microsurgery (American Association of Gynecologic Laparoscopists), *Washington DC*. J. M. Phillips, MD, 11239 S. Lakewood Blvd., Downey CA 90241.

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ORAL Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins,* i.e., *dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated).

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia. In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Dosage: Individualize for maximum beneficial effect.

ORAL *Adults:* Anxiety disorders, relief of symptoms of anxiety—Valium (diazepam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose.

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months).

INJECTABLE Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.)

For dosages in infants and children see below; have resuscitative facilities available.

I.M. use: by deep injection into the muscle.

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., *dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children *administer I.V. slowly;* for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed. Respiratory assistance should be available.

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful.

In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value.

How Supplied:

ORAL Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules—15 mg (yellow and blue), bottles of 100; Prescription Paks of 30.

INJECTABLE Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative.

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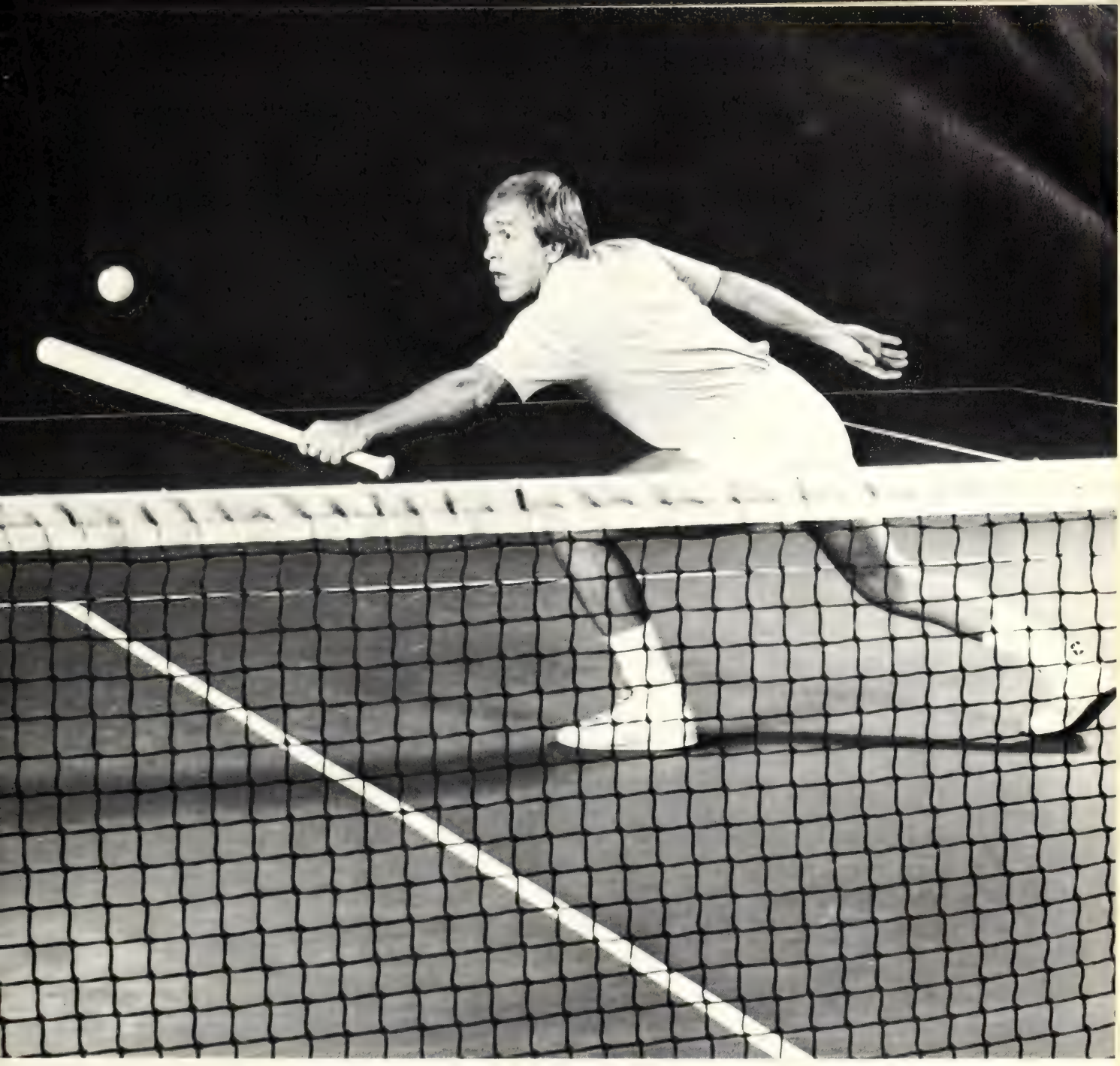

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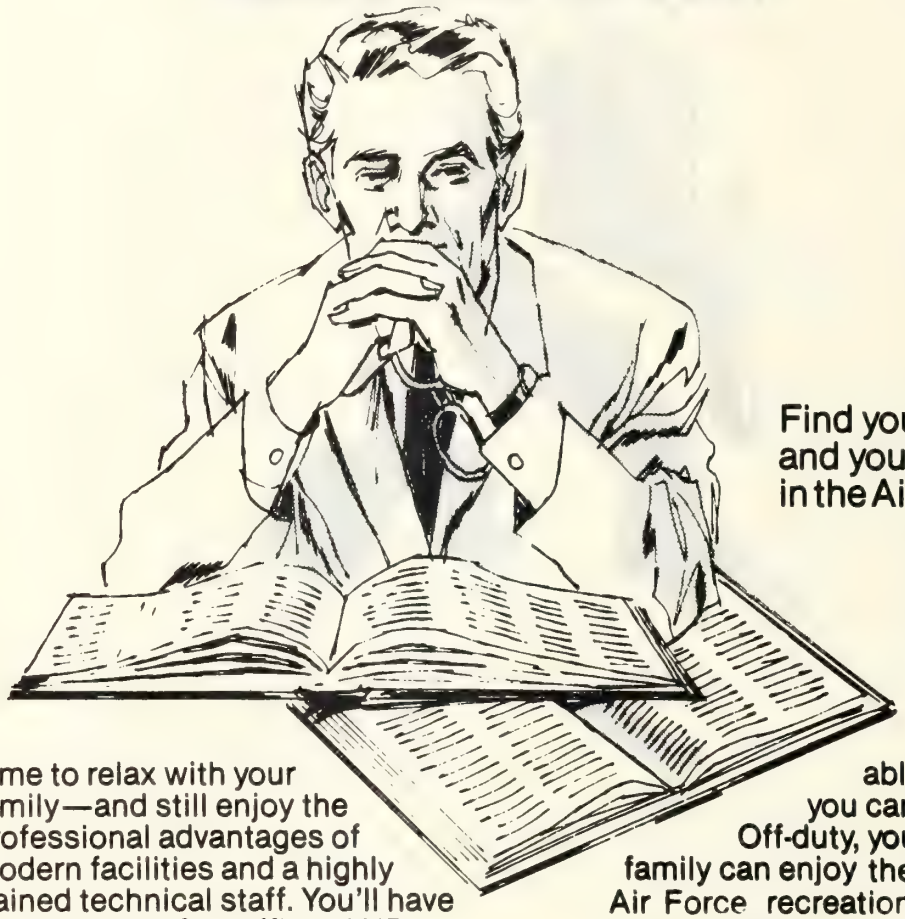

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On Being a Doctor

W. T. Thompson, Jr., MD, *Richmond, Virginia*

"I like the summing up that is ages old: The doctor's job is to cure rarely, to relieve sometimes, and to comfort always. In spite of all the changes medicine has undergone, this is still true with but minor revision: In 1983, he can cure often, not just rarely, and he can relieve pain and suffering almost always. His requirement to comfort is a constant."

SOME YEARS AGO an Alpha Omega Alpha lecture I gave was concerned with the physiologic aspects of oxygen therapy. After due respect was paid the AOA candidates, my talk was replete with slides showing the oxygen-carrying capacity of blood, the oxyhemoglobin dissociation curves, the effect of hyperbaric pressures, etc. Today I have no slides at all and am turning to philosophy rather than physiology, as is evident from my title.

This title appeals to me because I like the present participle as a verb form. It expresses action in an ongoing way—being, becoming, doing. Being a doctor is a continuing, never completed endeavor, one of constantly changing knowledge, skills, concepts and attitudes. You are always in the process of being a doctor.

Presented as the Kinloch Nelson Lecture on Student Honors Day, May 5, 1983, at the Medical College of Virginia/Virginia Commonwealth University at the time of the induction of Alpha Omega Alpha Scholars. An MCV graduate and AOA scholar himself, Dr. Thompson became chairman of his alma mater's Department of Medicine and is now Professor Emeritus. He is a former Editor of VIRGINIA MEDICAL.

Let me develop this thesis by posing to each of you three questions:

FIRST: What set you on this long, often rocky road to being a doctor? What really are your goals, your purposes? What motivates you, starts your wheels turning? This question of goals and motives demands firm answers when you are "being a doctor."

George Washington considered that everyone was motivated by two basic forces: "Interest," i.e., personal advantage, and "Honor," i.e., recognition by neighbors and country as being a worthy person.

Present day psychologists have expanded these to the four "P's" of Prestige, Possessions, Pride and Power. I hope that each of these in some measure will come to each of you. Self-interest is not an unworthy motive. But if it becomes distorted, if pride, prestige, possessions and power become goals in themselves, the primary or sole motivating forces in your professional life, you will be doomed to unhappiness and you and your chosen profession will be the poorer regardless of your fame or material success. Altruism alone cannot sustain you, but neither can Mammon.

Let me urge you to add a fifth motivating "P," Professionalism, i.e., standards, status, methods, actions and character in keeping with the learned profession that you have chosen for your way of life, for your vocation; a profession which throughout recorded history has been recognized for its service to others. In spite of technological advances and societal changes, medicine must remain essentially what it has always been, a profession, not a trade, with its own ends, means and intrinsic norms of conduct.

The Hippocratic Oath that you will take at graduation may seem outdated in some respects, hardly surprising after some 2500 years, and today we would hardly swear by Apollo physician, by Asclepius, by Health, by Panacea, and by all gods and goddesses. But listen to this: "I will use treatment to help the sick according to my ability and judgment," and "I will keep pure and holy both my life and my art," and "May I gain forever reputation among all men for my life and for my art."

The preamble of the Principles of Ethics developed by the American Medical Association states: "The medical profession has long subscribed to a body of ethical statements developed primarily for the benefit of the patient. As a member of this profession, a physician must recognize responsibility not only to patients, but also to society, to other health professionals and to self." Then follow seven specific items, the first of which is: "A physician shall be dedicated to providing competent medical service with compassion and respect for human dignity."

To say that medicine as a profession is under attack from many directions in our troubled and changing times is so self-evident that it hardly needs remarking. Some of it is our doing. All too many doctors have failed to hold to the high standards which are essential to our profession if it is to continue as we now know it. Some of it is due to the prevailing egalitarianism, leveling processes, social and legal activism. Read this challenge from the 1981 *Maryland Law Review*: "The current antitrust attack on professional power seems to provide most of the tools necessary to break down excessive public and private deference to the medical profession. . . . By exposing the hollowness of the profession's claims to special virtue, antitrust activity will contribute to the spreading perception of medicine as just another interest group scrambling for a place at the public trough."[1]

Listen to an entrepreneur who, at a recent meeting, told a group of physicians: "Whatever you physicians do to compete in the marketplace, make sure you don't get hung up on the traditional medical jargon of the physician-patient relationship and independent medical practice and the quality of care. Remember this: whatever you do, be sure to get your market share."[2]

Statements and thoughts such as these are revolting, really, and even terrifying. Imagine our profession, our practice of medicine, being described in terms of scrambling for a place at the public trough, and getting your market share.

My crystal ball becomes opaque, not just cloudy, when I try to look into the future and see what is in store for our profession. Heaven knows what the practice of medicine will be like when many of you open your offices.

But certain verities will surely obtain. The way one practices medicine will always be determined by the reasons one chose to be a doctor. Those motivated by the easy buck, by greed, by power will confirm, justify and accelerate the present attacks. Those motivated by professionalism, by their high ethical standards which stem therefrom, will offer the one hope of a reversal of these trends which portend such evil for the public as well as for the doctor.

The choice is up to each of you, and to each of your fellow physicians. The future of medicine depends upon your choice, and it is a continuing choice that must constantly be reaffirmed.

SECOND: How good are you at dealing with stress, your own as well as that in others? Abundant evidence shows that the doctor's job is a stressful one. Stress is undoubtedly a causative factor in the increasing numbers of impaired physicians, comprising perhaps as many as 10% of practicing doctors. Indeed, The Medical Society of Virginia has recently established the Physician's Health and Effectiveness Committee charged to develop means of prevention and rehabilitation.

Doctors are people, patients are people, all those with whom doctors have to deal are people. People have stress and people cause stress. People can be fun and funny, and wonderful, heart-warming, supportive. But people can be tough, very tough. People can be difficult, demanding, mean. People often do not see eye to eye with you, the doctor—or with each other, for that matter.

Doctors are subject to the same stresses and strains as every other member of society. Sometimes these can seem pretty great. Thoreau evidently had concluded this when he wrote, "The mass of men lead lives of quiet desperation." Unfortunately in this era the desperation is not always quiet.

Additionally, the nature of the doctor's job requires that he be in situations that are almost always very meaningful and often difficult. In matters of sickness or health, of life or death, success can bring great joy, failure, great sorrow.

Then there's the matter of fatigue, volume of work, pressure of time and such. I don't have to tell you, for you already know, that days can grow long and nights short, and may not contain enough hours for you to do what must be done. Ironically, sometimes the practice of medicine may be feast or famine. So many patients may be knocking at your office door that you can hardly stand it; but if only the wolf is at the door, that may be even more trying.

Finally, the prevailing condition of human affairs may confound what you have to do. I am thinking of such things as our litigious society and necessity to practice defensive medicine; of wishful thinking and over-selling of wondrous drugs, methods and techniques, with building up of high expectations doomed all too often to tragic disappointment; of quacks and crackpots, dope addicts, Baron Munchausens who involve you in their lives and problems by finding your name and telephone number in the Yellow Pages. The fact is that you are at the service—I started to say mercy—of anyone who dials your number, and this is both bane and blessing.

Stress has value. Shakespeare, the master student of human nature knew this, for he had Duke Senior declare in *As You Like It*: "Sweet are the uses of adversity, which, like the toad, ugly and venomous, wears yet a precious jewel in its head." It mobilizes energy, strengthens resolve, sustains action, and may be a major component of pleasure, excitement, fun; the exhilaration of striving, the satisfaction of success.

Stress puts you on your mettle, is a factor in relating successfully to others, in learning new facts and skills, in doing the job, and in achieving a reasonable adjustment to the situations you face, with, thereby, a diminution in the level of the stress. In other words, stress has inherent in it the potential for the reduction of the level of stress, or, even, its dissipation. This was aptly put when the interlocutor in the play, *Tea House of the August Moon*, declared, "Pain [stress] leads to thinking, thinking leads to wisdom, and wisdom makes this life more bearable."

Since stress is a basic part of being alive, and, more particularly, of being a doctor, learn to recognize it, its origins and manifestations in you. Make friends with it. Use it. Disarm it if you can, for it has the potential for great harm. It can demean, depress, break if the flesh is too weak, the level of stress too great, or mechanisms for dealing with it ill-chosen or inadequate.

Be aware of and exert control to the extent possible over unhealthy responses to stress. Psychological mechanisms of rationalization, projection, denial, withdrawal can be devastating if not recognized as such; and the turning to drugs and alcohol is a serious problem for an increasing number of doctors as we have noted. Substitute health-advancing activities such as useful hobbies, sports, a search for beauty in nature, art, music and literature. When all else fails, you may have to say with the Scottish chieftain, "I'm a little wounded, but I am not slain, I will lay me down for to bleed awhile, then I'll rise and fight with you again."

The internship should be the most productive, the finest year that you will ever spend—but it's no rose garden. The thorns may be very many and the blossoms seem very few at times. Indeed, during your intern days you may reach your nadir as a doctor, your very lowest point. I did. It came during the first several months, and I learned so much from it that I'm going to tell you about it.

To begin with, all interns—all doctors, and all people, for that matter—have a deep pool of paranoia that can well up to near-overflowing at times. The intern is, or considers himself to be, at the bottom of the pecking order, the one who is imposed upon the most, and the one who is subject to the beck and call of everyone with the possible exception of the elevator operator, and all too often that worthy keeps him waiting interminably when he is in the biggest hurry. (At least that was the way it was in my day, or seemed. The fact is, it isn't that way at all. The intern is a very special person indeed. Always remember that.)

The first three months of my 18-month-straight medical internship in my high post of Junior, or House Pup as it was also called, I was charged with doing all of the lab work-literally dozens of complete blood counts and urines each day—worked every other night and weekend, for which I was given room, board and laundry. About 10 o'clock one night, an elderly, flabby, uncommunicative, obese woman with long, unkempt, stringy, gray-white hair was admitted to our service. She had the most extreme pallor I had ever seen, and her forceful heart action with bounding pulse and labored breathing proclaimed mobilization of all resources in what appeared to be a failing attempt to maintain some degree of tissue oxygenation in the face of a very low hemoglobin.

It seemed to me that she needed blood, and sure enough, the Senior, a year along in his internship and my taskmaster, ordered a transfusion. My heart sank, for a transfusion was one of the most demanding, time-consuming and taxing of tasks for the Pup. There was no blood bank, and the typing lab closed in the afternoon. I had to do it all.

First I sent out a call for family members willing to give blood to come at once. Then, while waiting for the donors, I collected glassware and rubber tubing, sharpened the needles, set up the donor and delivery sets, and put it all in a half-filled pot of water and set it on the laboratory stove to sterilize by boiling. A number of distraught and anxious members of her family piled into the lab after an hour or so, and the tedious business of typing and direct cross-matching was begun. Fortunately, one of the family members who had come in proved to be compatible, and I drew 500 ml of his blood by gravity flow.

It was well after midnight when I started the blood going—whole blood, for there was no way in 1938 to give packed red blood cells. I sat by her bed to be sure that the blood was running in by slow drip in hopes of avoiding circulatory overload and to call for help promptly if heart failure developed. About 1 AM she roused a bit, opened her eyes, looked at me, and said, "Are you a doctor?" I replied in the affirmative, rather gruffly, I'm afraid, for I was very tired and sleepy, other patients had been admitted to the service who had to have lab work done before the night was out, and my paranoid level was rising. She then remarked, "You are only an intern, aren't you?" My even gruffer reply was, "Who else would be doing this at this time of night?" She then smiled at me in a kind and sweet way that lighted up her pale, plain face, and said, "Thank you, doctor," and went fast to sleep.

While sitting at her bedside with my fingers on her pulse, watching the slow drip of blood, and, yes, feeling sorry for myself, I began to realize in a specific, useful, practical way—not the intellectual recognition I had as a medical student—what medicine is all about. She wasn't a frowzy old woman who had waited until late evening to come to the hospital to add to my troubles. She was a fellow human being in need of the help that I, by virtue of my schooling and training in the profession that I had chosen, could give her. And this was but the prototype of what would be many other experiences and many other individuals.

This incident took place on the Fourth Medical Service of The Boston City Hospital on Ward Peabody 2, named for Dr. Francis W. Peabody, a brilliant scientist, investigator, and clinician, professor of medicine at Harvard Medical School, the first director of the great Thorndike Memorial Laboratory, and physician-in-chief of the Fourth Medical Service. Dr. Peabody died of a gstric malignancy at the height of his powers aged 46 in October 1927. In January 1927 he had delivered the George W. Gay Lecture at Harvard Medical School with the title, "The Care of the Patient." When I read it years later—I wish someone had told me about it when I was beginning my internship—the experience I had on Peabody 2 came into sharp focus. Listen: "What is spoken of as a 'clinical picture' is not just a photograph of a man sick in bed; it is an impressionistic painting of the patient surrounded by his home, his work, his relations, his friends, his joys, sorrows, hopes, and fears."[3] He concluded his lecture, "One of the essential qualities of the clinician is interest in humanity, for the secret of the care of the patient is in caring for the patient."[3]

Not many experiences are as clear-cut as mine. Not all patients are receptive and appreciative. Some are so driven by inner turmoil or fear or are in such pain or are so wrapped up in their illnesses and themselves that they have no time for anything or anybody, not even for the doctor. But they all, unlikely as it may seem at the time, are people, fellow humans, in need. The sooner that each of us understands and truly accepts this, the better off each of us is. This is the essence of dealing constructively with stress in our patients and in ourselves, which, in turn, is an essential component of being a doctor.

THIRD: What is the doctor's job? What does he do? He does many things and different things, depending upon his specific career choices, but, simply put, it all boils down to maintaining of health and treating the sick. The investigator has the added opportunity of increasing the body of knowledge and skills that contribute to maintaining health and treating the sick; and this includes so-called pure, or basic, research.

There may be a problem with definitions here, for the terms health and disease are subject to many interpretations. The early Greek physician, Galen, defined health in a rather bleak way as, "that condition in which we do not suffer, and are not impeded in the activities of life." The World Health Organization in its now-famous definition of "positive health" incorporated a kind of primeval euphoria: "Health is a state of complete physical, mental and social well-being, and not merely the absence of infirmity."

Expansive concepts of health can render the term almost meaningless and require ministrations that range far afield from the commonly accepted health requirements. Have you ever known anyone who enjoyed, or have you yourself ever experienced, complete physical, mental and social well-being other than for fleeting moments? Furthermore, health cannot be eternal, for death is the inevitable consequence of living.

A doctor has to establish appropriate goals for each patient and for himself as well. But let me emphasize the word appropriate. It must be his best considered judgment reached only after a hard look at all of the facts and may include supporting opinions. While a goal that is too high for you or for your patients may cause anxiety, one that is needlessly low may be equally or more distressing and wasteful, especially if it is the result of ignorance, neglect, laziness or lack of concern. Above all else, a doctor must be competent—competent to make proper decisions and to deal effectively with disease processes and with the sick person.

The doctor accomplishes his mission by achieving a proper blending of the art, science and technology of medicine. For a perceptive and experienced physician, an adversarial relationship never exists between art, science and technology. One or another might require emphasis and particular attention depending upon the circumstance. Keep in mind always that the doctor of medicine is the only member of our society who is trained to deal with the diseases of metabolic derangements, malignancies, infections, organ failures and such. There is no one else to do it. At times he may have to focus on science and technology to the exclusion of all else.

Dr. Lewis Thomas, who has impeccable credentials as a scientist, has written of this in his most recent book, *The Youngest Science:* "If I develop the signs and symptoms of malignant hypertension, or cancer of the colon, or subacute bacterial endocarditis, I want as much comfort and friendship as I can find at hand, but mostly I want to be treated quickly and effectively so as to survive, if that is possible."[4] But note that he spoke of signs and symptoms, of physical examination and history-taking, of findings derived from a study of the patient by the doctor.

Dr. Peabody wrote of this relationship also, putting the art, science and technology of medicine in true perspective: "The treatment of disease may be entirely impersonal; the care of the patient must be completely personal. The significance of the intimate personal relationship between physician and patient cannot be too strongly emphasized, for in a large number of cases, diagnosis and treatment are directly dependent upon it."[3]

Automated laboratory testing, computed axial tomography, positron emission tomography, nuclear magnetic resonance, new parts for old, machines for organs, computerized medicine—all of these and many more marvels of science and technology are here today, with more promised for tomorrow. There is no turning back, and no one in his right mind would want to. Sick people can be diagnosed and treated in a way and with an accuracy and cure rate that could not even be imagined just a few years ago.

But these changes, grand and spectacular as they may be, come at a price in human relationships that may be too high unless conscious effort is made to maintain the contributions of the art of medicine, the relationship of doctor and patient.

Says Dr. Thomas: "This uniquely subtle relationship has roots that go back into the beginnings of medicine's history and needs preserving. To do it right has never been easy; it takes the best of doctors, the best of friends. Once lost, even for as short a time as one generation, it may be too difficult a task to bring it back again.

"If I were a medical student or an intern, just getting ready to begin, I would be more worried about this aspect of my future than anything else. I would be apprehensive that my real job, caring for sick people, might soon be taken away, leaving me with the quite different occupation of looking after machines. I would be trying to figure out ways to keep this from happening."[4]

I like the summing up of the doctor's job that is ages old: The doctor's job is to cure rarely, to relieve sometimes, and to comfort always. In spite of all the changes that medicine has undergone, this is still true with the altering of only two modifying adverbs. In 1983 he can cure often, not just rarely—indeed, many of the diseases that plagued this world of ours have now disappeared—and he can relieve pain and suffering almost always. His requirement to comfort is a constant.

I wish each of you a lifetime commitment to being a doctor and to honoring your chosen profession.

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Parathyroid Crisis in Secondary Hyperparathyroidism

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There are few reports of parathyroid crisis due to secondary, rather than primary, hyperparathyroidism, but the authors describe two patients, both with chronic renal failure, whom they treated for this acute state. A review of the literature provides a context for these cases.

In 1965, Payne and Fitchett[1] presented a review of hyperparathyroid crisis, accumulating 68 cases in the world literature and adding two more cases. Since that report, at least 90 additional cases have been described in the literature.[2-13] Nearly all of these cases have occurred in patients with *primary* hyperparathyroidism, with the majority of these due to parathyroid adenomas (85%). All reports have emphasized that early surgical intervention is critical.

Secondary hyperparathyroidism is an almost inevitable complication in patients with end-stage renal failure who are maintained for long periods by dialysis. Most of these patients can be managed by nonoperative measures. However, parathyroidectomy may be necessitated by the development of certain severe manifestations of secondary hyperparathyroidism, including renal osteodystrophy, persistent hypercalcemia, intractable pruritus, and metastatic calcifications. An additional indication for surgery in patients with secondary hyperparathyroidism is hyperparathyroid crisis. Cases of hyperparathyroid crisis associated with *secondary* hyperparathyroidism have rarely been reported in the literature, but we recently treated two such patients, who form the basis of this report. These cases emphasize the importance of vigorous preoperative management and urgent parathyroidectomy.

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Case Reports

Case 1.

A 36-year-old black male with end-stage renal failure secondary to hypertensive nephrosclerosis was admitted to Medical Center Hospitals, Norfolk General Division, on 9/6/79 with a 48-hour history of mid-abdominal pain, nausea and vomiting. There was no past history of similar symptoms. The patient had been on chronic hemodialysis for the preceding three years. During that period, he had received two cadaveric kidney transplants (2/1/77 and 6/7/79), both of which were rejected within one month. Multiple serum calcium levels had ranged between 8.1 and 10.8 mg/100 ml (Fig. 1).

Physical examination revealed a chronically ill black male in moderate distress, complaining of abdominal pain. Temperature was 97.8°F. Abdominal examination demonstrated tenderness in the mid-epigastrium radiating to both flanks. Mild rebound tenderness was present, but no guarding was noted. No masses or organomegaly were appreciated. Bowel sounds were normal.

Initial laboratory studies revealed a serum calcium of 14.0 mg/100 ml and phosphorus of 4.3 mg/100 ml. WBC was 11,400/cu mm with a left-shift. Electrolytes were within normal limits. Serum amylase was markedly elevated to 2,230 units/dl. Abdominal X-rays were unremarkable. A diagnosis of acute pancreatitis was made.

On the morning following admission, the patient experienced a grand mal seizure, which was controlled with intravenous phenytoin sodium (Dilantin®). The patient experienced two more seizures in the hemodialysis unit. Following dialysis, the patient remained in a post-ictal state with stupor and semi-purposeful response to painful stimuli. A spinal tap was unremarkable. Pre-dialysis serum calcium was 14.1 mg/100 ml but was reduced to 11.0 mg/100 ml after a four-hour dialysis. A parathyroid hormone level drawn at this time was markedly elevated to 2,196 pg/ml.

Surgical consultation was then obtained, and a diagnosis was made of hyperparathyroid crisis due to secondary hyperparathyroidism. An emergency subtotal parathyroidectomy was performed, with one-third of the right inferior gland preserved. The histologic diagnosis was secondary parathyroid hyperplasia. The serum calcium several hours after surgery was 10.1 mg/100 ml and ranged between 7.3 to 9.9 over the next week.

Postoperatively, the patient's neurological status gradually improved over the ensuing two weeks, although his global aphasia was slow to resolve. His abdominal pain subsided and the serum amylase returned to normal. He was discharged 9/24/79 with a normal speech pattern and a serum calcium of 8.9 mg/100 ml.

Case 2.

A 50-year-old black male with end-stage renal failure secondary to hypertensive nephrosclerosis was admitted to Medical Center Hospitals, Leigh Memorial Division, on 1/25/80 with a three-month history of progressive weakness and nervousness. He described the gradual onset of weakness, especially on the left side, accompanied by headache, depression, confusion, weight loss and tremulousness of the hands. Due to the marked change in his

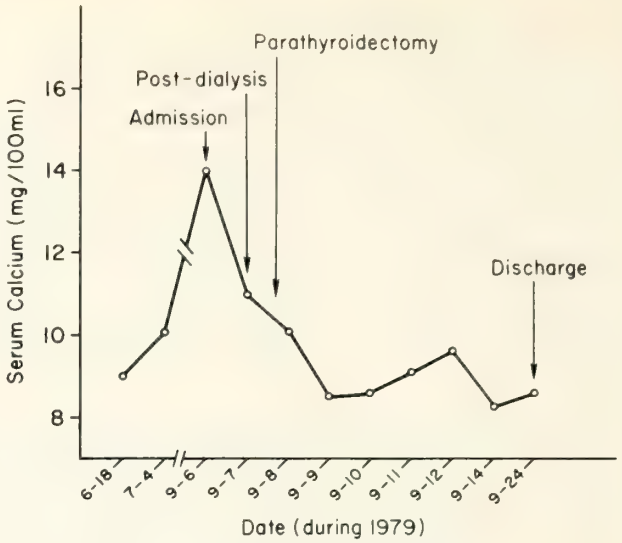

Fig. 1. Case 1, serum calcium levels.

clinical condition, he was admitted for further evaluation of his neurological status.

The patient had been started on hemodialysis in December 1977. A kidney transplant in October 1978 was rejected and removed the following month. Since that time he had been maintained on chronic hemodialysis. During the past year his serum calcium levels had been gradually increasing to a range of 12.0 to 14.0 mg/100 ml (Fig. 2), and his parathyroid hormone level was noted to be elevated on two occasions.

Physical examination revealed a chronically ill black male in no acute distress. He was somewhat slow in his response to questions. There was generalized weakness, worse on the left side. Rapid finger movements were performed slowly bilaterally with some tremor on finger-to-nose testing. He

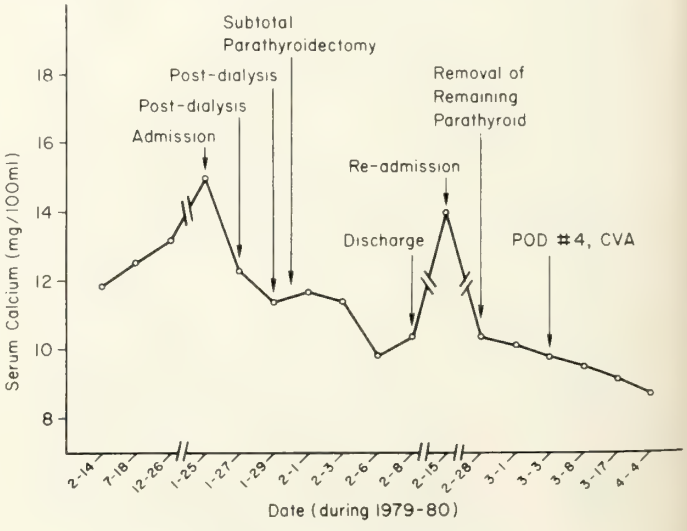

Fig. 2. Case 2, serum calcium levels.

walked with a wide-based gait. No carotid bruits were noted.

Admission laboratory studies revealed a serum calcium of 15.0 mg/100 ml, phosphorus of 2.2 mg/100 ml, creatinine of 13.4 mg/100 ml, and BUN of 33 mg/100 ml. On the basis of his progressive CNS symptoms and his markedly elevated calcium level, a diagnosis was made of hyperparathyroid crisis due to secondary hyperparathyroidism. Hemodialysis was successful in reducing his serum calcium to 12.3 mg/100 ml. Another dialysis treatment on 1/29/80 further reduced the calcium to 11.4 mg/100 ml. A parathyroid hormone level drawn at this time was elevated to 313 pg/ml.

Satisfactory response to hemodialysis and subsequent improvement in the patient's condition allowed a short delay in his operative management. He was taken to the operating room on 1/31/80, at which time he underwent a subtotal parathyroidectomy, with one-third of the right superior gland preserved. The histologic diagnosis was secondary parathyroid hyperplasia. He had an uncomplicated postoperative course, although his serum calcium levels were slow to fall, the lowest value being 9.8 mg/100 ml.

The patient's CNS symptoms resolved, but shortly after discharge he experienced severe bone pain in his left lower extremity, which required readmission on 2/15/80. The serum calcium level at that time was 14.0 mg/100 ml. Bilateral inferior thyroid angiograms and selective venous sampling failed to localize definitely an area of persistent hyperfunctioning parathyroid tissue. A second exploration of the neck was performed on 2/28/80, at which time the only parathyroid tissue identified was the small portion of the preserved right superior gland. This remaining tissue was removed, as was the left lobe of the thyroid gland.

Postoperatively, the patient's calcium levels promptly decreased to the range of 8.6 to 10.4 mg/100 ml. Unfortunately, he experienced a cerebrovascular hemorrhage on the fourth postoperative day and died on 4/20/80.

Discussion

Secondary hyperparathyroidism (HPT) is almost universal among patients with chronic renal failue. It develops as renal function deteriorates and the kidney loses its ability to excrete phosphate. The resultant hyperphosphatemia produces a reciprocal decrease in the serum calcium level, which, in turn, stimulates a compensatory secretion of parathyroid hormone. Abnormal vitamin D metabolism also contributes to the development of secondary HPT by means of decreased intestinal uptake of calcium. In an attempt to maintain normal serum calcium levels, the increased secretion of parathyroid hormone results in increased calcium mobilization from bone with skeletal demineralization, which may lead to renal osteodystrophy.

In uncomplicated secondary HPT, the serum calcium usually does not rise above a normal level. However, in the late stages of the disease, the parathyroid glands gain a measure of autonomy and fail to respond to the usual feedback mechanism, with a resultant elevation of the serum calcium levels. (Although some authors refer to this phenomenon as "tertiary hyperparathyroidism," we reserve this term for the autonomous parathyroid hyperfunction that develops following successful renal transplantation.) This hypercalcemia, in addition to continued retention of phosphate by the impaired kidneys, results in an increase of the calcium-phosphate product and thus a precipitation of calcium salts in metastatic locations (arteries, muscle, dermis, etc.).

Symptomatic secondary HPT is often controlled in chronic hemodialysis patients by the early institution of appropriate medical management of calcium and phosphorus metabolism.[14-16] Normal serum calcium and phorphorus levels may be approached by a therapeutic regimen that includes high calcium intake, phosphate restriction, oral phosphate-binding antacids, vitamin D supplements, 1,25-dihydroxy-cholecalciferol, and careful adjustment of calcium and magnesium in the dialysis fluid. In addition, cimetidine has recently been shown to decrease circulating levels of parathyroid hormone in patients with HPT.[17]

Most patients with chronic renal failure can be managed in this manner. However, parathyroidectomy must be considered when medical therapy is unsuccessful and severe manifestations of secondary HPT are exhibited.[14,18-23] Indications for surgery include renal osteodystrophy (severe bone pain, pathologic fractures, etc.), persistent and symptomatic hypercalcemia, intractable pruritus, and metastatic calcifications (intravascular and soft tissue).

The two patients in this report, whose severe secondary hyperparathyroidism was complicated by hyperparathyroid crisis, add another indication for surgical treatment.

The various published series of chronic renal failure patients requiring parathyroidectomy undoubtedly include certain cases of hyperparathyroid crisis that are not specified as such. For

example, Gordon et al[20] reported two patients with secondary hyperparathyroidism who developed symptomatic hypercalcemia. One 36-year-old patient experienced lethargy, mental confusion, anorexia and nausea in association with a serum calcium level of 13.0 mg/100 ml. Following a neck exploration and removal of two parathyroids, he became asymptomatic and normocalcemic.

Bradley and Wells[22] described three patients with secondary hyperparathyroidism in whom the serum calcium approached the level seen in hyperparathyroid crisis. A subtotal parathyroidectomy was performed, and in each case the hypercalcemia was promptly reduced to an acceptable range.

Review of the nephrology literature reveals a case report by Ball et al[24] in which a 56-year-old white female with end-stage renal failure developed generalized seizures and deteriorating neurological status, a syndrome generally described as "dialysis dementia." Elevated serum calcium levels in the range of 10.5 to 12.5 mg/100 ml were noted, and a subtotal parathyroidectomy was peformed. Postoperatively, her neurological symptoms resolved and the serum calcium fell to 7.5 mg/100 ml. Although the etiology of dialysis dementia is obscure, the dramatic improvement of this patient following parathyroidectomy suggests that secondary hyperparathyroidism contributed to her clinical picture.

Other chronic renal failure patients with progressive CNS symptoms may satisfy the criteria for hyperparathyroid crisis. Another case report by Ball et al[25] described a 24-year-old white male on chronic hemodialysis who developed involuntary muscle movements, nystagmus, confusion and an elevated serum calcium of 13.8 mg/100 ml, with dramatic improvement following subtotal parathyroidectomy. Sivula et al[26] reported on five chronic renal failure patients in whom mental disturbances were improved with subtotal parathyroidectomy.

The definitive treatment of hyperparathyroid crisis is surgical removal of the hyperfunctioning parathyroid tissue. In primary hyperparathyroidism, when an adenoma is responsible for the crisis, removal of the adenoma provides an effective cure. However, secondary hyperparathyroidism produced by hyperplasia of all parathyroid glands requires a subtotal parathyroidectomy, with no more than the equivalent of one-half of a normal gland preserved. Total parathyroidectomy may be employed in order to avoid residual hyperparathyroidism (a complication of Case 2), but this technique should not be performed on patients who are candidates for renal transplantation, since post-transplant hypoparathyroidism will most likely result.[27] Total parathyroidectomy and autotransplantation of small amounts of diced parathyroid tissue into the muscles of the forearm as described by Wells et al[28] may answer this problem.

Hyperparathyroid crisis is almost invariably fatal in those patients who do not undergo surgical therapy. The overall mortality ranges between 50%-65%. The mortality of those who undergo parathyroidectomy may be as high as 24%,[4] although in a large series by Schweitzer et al,[7] the mortality was only 6.8%.

Summary

This report suggests that hyperparathyroid crisis is an infrequent complication of secondary hyperparathyroidism and may have a clinical presentation similar to crisis associated with primary hyperparathyroidism. The diagnosis may not be as easily recognized, in patients with advanced renal disease, however, due to the presence of coexisting problems which can mask the symptom complex. The criteria for hyperparathyroid crisis in secondary hyperparathyroidism are 1) an elevated serum calcium level, usually greater than 13.0 mg/100 ml but not necessarily as high as that seen in primary hyperparathyroid crisis; and 2) rapid changes in the general condition of the renal failure patient, with central nervous system symptoms often dominating the clinical picture. Successful treatment requires prompt recognition, vigorous preoperative control of the hypercalcemia, and expeditious surgical management by parathyroidectomy.

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In vitro studies demonstrate

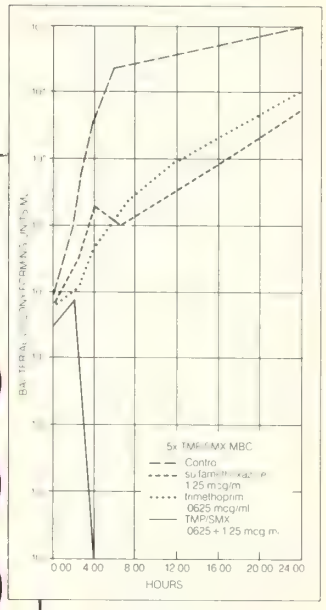
Bactericidal activity with minimal resistance

Percent of isolates of common uropathogens sensitive to BACTRIM and to other antimicrobials

	Escherichia coli	*Klebsiella pneumoniae*	*Proteus mirabilis*	*Proteus vulgaris*	*Proteus sp*	*Enterobacter sp*	*Enterobacter aerogenes*	*Enterobacter cloacae*
BACTRIM	**96%** 328,598	**89%** 63,551	**93%** 62,691	**84%** 4174	**91%** 4381	**88%** 1736	**96%** 7326	**92%** 11,856
ampicillin	72% 345,209	5% 65,466	85% 67,338	16% 4381	70% 4402	10% 1775	9% 7759	11% 12,091
cephalexin†	81% 343,339	85% 67,031	92% 67,027	16% 4398	80% 4365	22% 1834	13% 7571	12% 11,846
nitrofurantoin	97% 307,785	65% 61,162	7% 59,775	13% 3848	14% 3059	67% 1707	60% 7109	67% 10,830

†Analogous to cephalothin, the primary antibiotic disc used in testing
Source: The Bacteriologic Report, BAC-DATA Medical Information Systems, Inc., Winter Series, 1981-82
Numbers under percentages refer to the projected number of isolates tested.

RAPID *IN VITRO* DESTRUCTION OF *E. COLI**



Kill curve kinetics of Bactrim and its individual components against *E. coli in vitro*.[1]

The bactericidal action of Bactrim has been demonstrated *in vitro* on laboratory strains of *E. coli*[1,2] and on clinical isolates of *E. coli, Klebsiella-Enterobacter, Proteus mirabilis* and *Morganella morganii*[3]—the most common causative organisms of urinary tract infections.[4] More than 100 published studies attest to the efficacy of Bactrim in recurrent urinary tract infections due to these organisms.[5] In comparative studies with other antimicrobials, Bactrim has consistently demonstrated unsurpassed efficacy during therapy.[6-11]

Resistance to Bactrim develops more slowly than to either of its components alone *in vitro*.* Among urinary tract isolates, resistance has rarely emerged in susceptible strains.[5,12] Bactrim is contraindicated in pregnancy at term, during lactation, in infants less than two months old and in documented megaloblastic anemia due to folate deficiency.

Initial episodes of uncomplicated urinary infections should be treated with a single-agent antimicrobial.

Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

b.i.d. *for recurrent urinary tract infections*

**In vitro* data do not necessarily predict clinical results.

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See next page for references and a summary of product information.

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Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii*. It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients. *Pregnancy:* Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies

Dosage: *Not recommended for infants less than two months of age.*

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN.

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets*, each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 20. *Tablets*, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension*, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension*, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per tea spoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint)

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BRIEF SUMMARY

PROCARDIA® (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina), PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%; palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request

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"I can do things that I couldn't do for 3 yrs. including joining the human race again."

Quotes from an unsolicited letter received by Pfizer from an angina patient.
While this patient's experience is representative of many unsolicited comments received, not all patients will respond to Procardia nor will they all respond to the same degree.

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"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA[*] *as soon as it became available. The change in my condition is remarkable."*

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks,[1] taking fewer nitroglycerin tablets,[2] doing more, and being more productive once again

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%)

for the varied faces of angina

* Procardia is indicated for the management of:
1) Confirmed vasospastic angina.
2) Angina where the clinical presentation suggests a possible vasospastic component.
3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

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Please see PROCARDIA *brief summary on adjoining page*

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Current Concepts in the Etiology and Pathology of Bell's Palsy

Francis H. McGovern, MD,
and Jose Estevez, MD,
Danville, Virginia

The authors describe good results in treating Bell's palsy by controlling rebound mast cell degranulation, which they suspect as the cause of facial canal edema leading to nerve damage. They document their treatment methods and review what is known of the etiology of the disease.

THE DIAGNOSIS of Bell's palsy is not a haphazard or random one. The disease is precisely defined as a unilateral or bilateral demyelinating peripheral facial palsy of sudden onset, occurring in an otherwise healthy person who on careful examination fails to show a cause for the paralysis.

Bell's palsy has an inherent tendency to recover spontaneously in 80% of patients. Fifteen percent recover partially and are left with a slightly impaired facial function. Five percent suffer the misfortune of a serious degree of permanent facial deformity.

Because so many patients with Bell's palsy can recover with no treatment, clinicians have adopted a laissez-faire attitude and often treat Bell's palsy with benign neglect. In reality, our efforts should be turned to the 20% of patients who fail to recover full facial function or develop associated movements.

From the Department of Otolaryngology, University of Virginia Medical Center and the Department of Pathology, Memorial Hospital, Danville. Address correspondence to Dr. McGovern at 139 South Main Street, Danville VA 24541.

Submitted 11-4-82.

Etiology

The precise etiology and pathogenesis of Bell's palsy is still unknown. Many investigators now believe that Bell's palsy is not a single disease, but a group of entities.

The popular hypothesis that Bell's palsy is of viral etiology, in which the ever present herpes simplex virus is usually incriminated, is certainly attractive. The neurotropic herpetic family of viruses is associated with viral polyneuritides, which are occasionally long-lasting and severe. Herpes zoster is often linked with facial nerve involvement, concomitant with significant dysfunction of other cranial and/or peripheral nerves, and is characterized by considerable pain. Facial palsy and infectious mononucleosis caused by Epstein-Barr virus, a member of the herpes family, has been described but is uncommon.

The viral polyneuritides are due to T-cell mediated macrophage activation, and demyelination is a late occurrence, if it occurs at all. Nerve dysfunction, paralysis and pain, however, develop quite early. The evidence supporting herpes simplex as the sole or main etiologic agent in Bell's palsy has been unconvincing; the multiplicity of viral antibodies and antigens reported by different investigators makes its role unclear. A recent quite thorough report on the histology of Bell's palsy says that the disease may represent the infiltrative neuritis characteristic of a herpesvirus group infection.[1]

Abramsky[2] found that lymphocytes from patients with Bell's palsy, like those in Guillain-Barré syndrome, undergo significant stimulation when cultured in the presence of myelin basic protein. This interesting finding may be secondary to demyelination rather than its cause. Autoimmunity to myelin basic protein is present in most cases of neural injury, whatever the cause.

A few facts are more or less agreed upon by most investigators: 1) The anatomical location of the facial nerve enveloped in its bony canal has a significant role in the paralysis of Bell's palsy. 2) Those who have examined a paralyzed facial nerve clinically and histopathologically have considered

edema to be the most salient pathological finding. 3) Antibodies to several viruses (not just a single one) have been found in a number, but not the majority, of patients developing Bell's palsy. 4) Histological accounts of the entity have shown that the majority of cases reveal an acellular edematous neuropathy.[3]

Clinical Trial

Our experimental model[4] of facial paralysis in dogs utilized several triggering mechanisms for intracanalicular edema production, all of which led to paralysis of different degrees and duration. In our latest model the edema is secondary to an Arthus reaction and is triggered by the injection of horse serum into the fallopian canal of a hyperimmunized animal. As a result of these experiments, we have developed the following concepts of the etiology and pathogenesis of Bell's palsy as immunologic in nature.

The edema of the Arthus reaction is brought about by massive mast cell degranulation initiated by release of complement components. Complement activation is caused by the formation of antigen antibody complexes. In most cases the Arthus reaction lasts only 24-36 hours and is self-limited.

Within the facial canal, however, the edema produces an increased intracanalicular pressure, and the pressure produces a rebound degranulation of the activated mast cells, perpetuating the edema. Sustained pressure causes paralysis and leads to nerve atrophy and demyelination.

The treatment of experimental animals with an induced facial paralysis by a mast cell degranulation inhibitor, cromolyn sodium,[5] greatly reduced the neural edema. There was a concomitant suppression of the paralysis and prevention of nerve degeneration.

As an experiment, these findings were applied clinically, using cromolyn sodium by inhalation. The patients were divided into two groups: Group 1 consisted of a) those patients who started the inhalations within five days after the onset of paralysis, and b) those who maintained the strict cromolyn schedule until recovery. Group 2 was comprised of a) those patients who were unable to maintain effective inhalations because of age, mental capacity or chronic obstructive pulmonary disease, and b) those who showed poor compliance or discontinued medication.

All of the 92 patients in Group 1 recovered completely within 16 days after the onset of paralysis. The 22 patients in Group 2 averaged 78% return of function after 37 days of treatment.[6]

It should be noted that if the patient is first seen ten days to two weeks after the onset of the disease, management of the facial paralysis is directed by the nerve excitability test. If the nerve responds actively in the range of 4-6 milliamperes, no treatment is indicated, and the patient will recover spontaneously.

If the nerve excitability response is lost, however, corticosteroid treatment should be started at high dosage. Progressive intracanalicular edema can be controlled and ameliorated by the nonspecific multiple immunological actions of corticosteroid therapy. Further nerve damage may be prevented and the course of the paralysis can be shortened. The starting dose of prednisone is 120 mg daily for 4-7 days, continuing for three weeks on a declining dosage schedule.

Currently we are carrying on experiments with lodoxamide, another mast cell degranulation inhibitor, apparently with similar results.

Discussion

It is quite evident that proposing a single etiologic pathway for Bell's palsy is not tenable. On the other hand, based on our experimental model and clinical experience, we can advance a single chain of pathogenetic events, leading to either total recovery or to variable permanent loss of nerve function: 1) Increased pressure within the canal causes paralysis. 2) Edema, due to mast cell degranulation, causes increased intracanalicular pressure. 3) Besides paralysis, increased intracanalicular pressure produces rebound mast cell degranulation, which perpetuates the edema and the increased pressure. 4) When there is a generalized immune reaction (such as in viral or bacterial diseases, or in allergic conditions) mast cells are more susceptible to nonspecific degranulation (mast cell activation). 5) Rebound degranulation due to increased intracanalicular pressure is more severe when mast cells are activated.

We believe Bell's palsy is initially caused by edema within the facial canal, be it in the facial nerve itself or the perineural tissues or both. This edema is likely due to an episode of mast cell degranulation.

It is possible that a neurotrophic infectious agent such as herpes simplex virus, involving several cranial nerves, may produce transient dysfunction in all of the cranial nerves. It is also possible that an immune reaction occurring within the facial canal can bring about the chain of events described in our experimental model. It is certainly a very similar situation. An active immune reaction to the virus must be taking place. Mast cell activation is proba-

bly present. An Arthus reaction occurring in any other nerve would produce transient changes of little clinical significance, but within the tight constraints of the facial canal, the rebound mast cell degranulation would perpetuate the intracanalicular pressure and produce the symptoms we diagnose as Bell's palsy.

Other agents, whether neurotropic or opportunistic, may trigger a similar sequence of events through similar mechanisms, and in a predisposed individual with certain genetic or immunologic idiosyncrasies, nonspecific mast cell degranulation may initiate the process, perhaps starting in the extracanalicular area of the nerve (such as the exposed portion of the chorda tympani).

Summary

Based on the concepts thus far proposed, we feel that successful treatment of Bell's palsy can be accomplished by controlling rebound mast cell degranulation, thereby preventing nerve damage. In the management of Bell's palsy in the stage of neuropraxia, we advise the use of a mast cell degranulation inhibitor, such as cromolyn sodium, which requires oral inhalation, an awkward method of treatment, but is unusually safe and has been used for many years in the prevention of asthma with few minor side effects. At present we are experimenting with other inhibitors that can be given orally.

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Severe Metabolic Complications in a Cross-Country Runner with Sickle Cell Trait. Kathy J. Helzlsouer, MD, Frederick G. Hayden, MD, and Alan D. Rogol, MD, *Charlottesville.*

A 17-year-old black male with sickle cell trait collapsed after completion of a 5,000-m cross-country race. He manifested CNS dysfunction, disseminated intravascular coagulation, rhabdomyolysis, renal insufficiency, and deep venous thrombosis. Sickle cell crisis may have been etiologic in the pathogenesis of this constellation of abnormalities. JAMA 249:777–779, 1983

Beta-Hemolytic Streptococcal Pharyngitis: Uncommon in Infectious Mononucleosis. Susan C. Merriam, RN, and Richard P. Kelling, MD, *Charlottesville.*

Infectious mononucleosis (IM) and β-hemolytic streptococcal pharyngitis may present similar clinical pictures and may occur concurrently; however, recent studies have differed sharply with older ones that cited high rates of concurrent disease. Our study of 100 consecutive new patients meeting strict criteria for a diagnosis of IM found only 4% with positive throat cultures for group A β-hemolytic streptococci at the time of diagnosis. Use of antibiotics for presumed streptococcal pharyngitis in patients with IM is unjustified. Only patients with positive cultures should be treated. South Med J 76:575–576, 1983

Posttraumatic Osteolysis of the Clavicle. Stephen F. Quinn, MD, and Ted A. Glass, MD, *Charlottesville.*

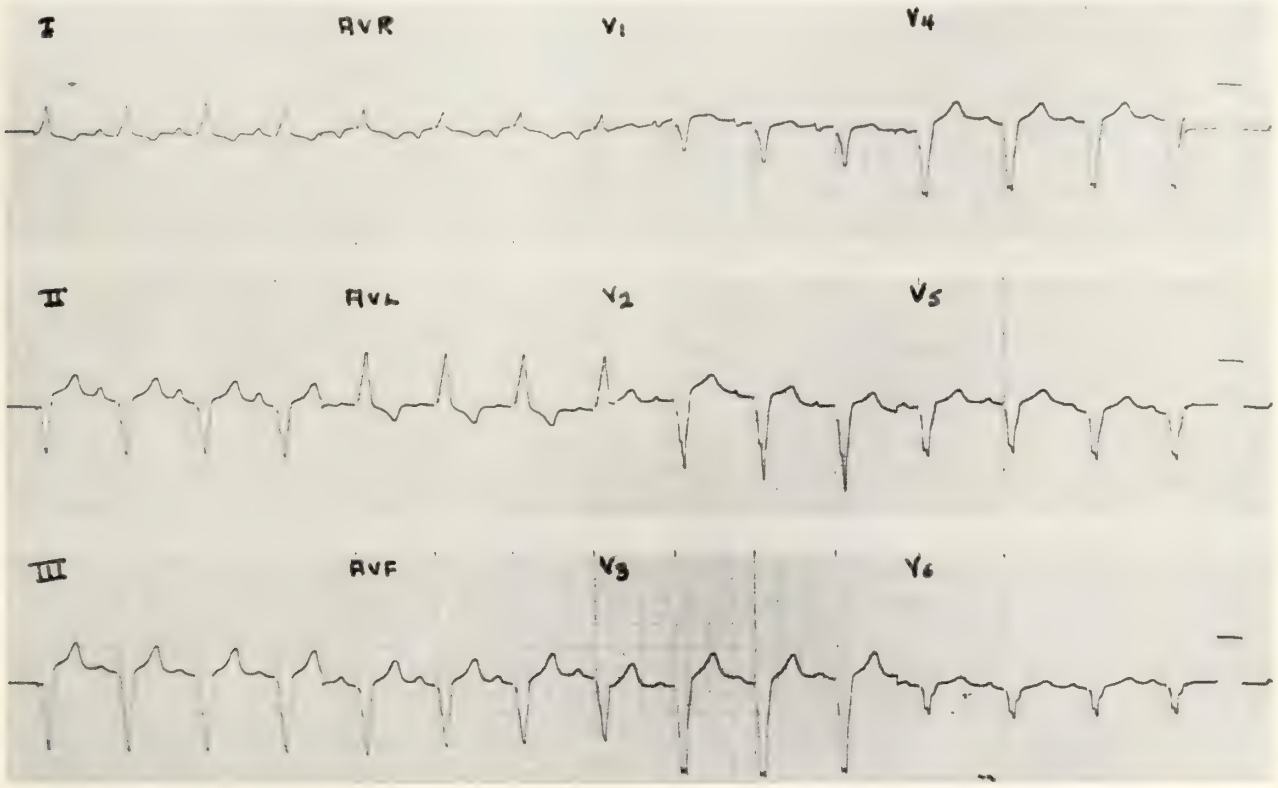
Posttraumatic osteolysis of the distal clavicle should be considered when continued pain follows trauma. The osteolysis and accompanying symptoms can be partially alleviated by immobilization of the shoulder joint. The pathology is poorly understood, but synovial hyperplasia appears to play an integral part in the resorptive process. South Med J 76:307–308, 1983

Can You Diagnose This?

A medical puzzle prepared by
Donald W. Romhilt, MD, and Margaret Doyle, RN, *Richmond, Virginia*

This 12-lead electrocardiogram, which was recorded with a three-channel machine, was taken from a 25-year-old woman with known congenital complete heart block. Her pacemaker had been replaced the day before. *From the Department of Medicine, Division of Cardiology, Medical College of Virginia/Virginia Commonwealth University.*

DIAGNOSIS/DISCUSSION NEXT PAGE



Diagnosis

Normal sinus rhythm followed by artificial ventricular pacemaker spike and QRS complex at a consistent interval after the P-wave, indicating an artificial pacemaker that is atrial-sensing and ventricular-pacing. It is one of the new generation of pacemakers, the DDD type, which is dual pacing (atrium and ventricle), dual sensing (atrium and ventricle), and triggered and inhibited.

Discussion

During the 1970s, most artificial pacemakers were demand ventricular pacemakers of the VVI or VVT type (ventricular pacing, ventricular sensing, and either triggered or inhibited). The characteristic EKG of these packemakers has a spike followed immediately by a wide QRS complex at a set rate unless a native beat occurs which is sensed by the pacemaker, and then the pacemaker does not initiate the impulse.

In an effort to preserve the atrial and ventricular sequence of contraction, AV sequential pacemakers, or DVI type (dual pacing, ventricular sensing, inhibited), were developed. Their characteristic EKG pattern has a pacemaker spike at a set rate, followed by an atrial contraction; then, at a consistent interval, a second spike is followed by a wide QRS, unless a native beat occurs which is sensed within the ventricle, and then the pacemaker does not initiate a ventricular impulse.

In addition, a P-wave synchronous pacemaker, or VAT type (ventricular pacing, atrial sensing, triggered), was developed. This pacemaker characteristically has an EKG with a native P-wave followed at a consistent interval by a spike and a wide QRS complex. It responds to increases and decreases in the native heart rate and is more physiologic in this respect than the set rate of an AV sequential pacemaker; it will not, however, sense premature ventricular contractions, increasing the likelihood of competitive rhythms. In an effort to eliminate competitive rhythms, a P-wave synchronous pacemaker of the VDD type (ventricular pacing, dual sensing, and triggered and inhibited) was put on the market.

A fully automatic pacemaker, or DDD type, has become available recently. This pacemaker preserves physiologic function and combines the pacing capability of previous units within the limits of lower and upper rates that can be programmed. It can stimulate 1) the atria only, in patients with sinus bradycardia and normal atrioventricular conductions; 2) the atria and ventricles sequentially, in patients with sinus bradycardia with atrioventricular block; 3) the ventricles only, after a preset atrioventricular interval upon sensing the native P-wave, in patients with sinus rhythm and atrioventricular block (when, to increase cardiac output, it is important to pace at the patient's intrinsic sinus rate, as in the patient described here); 4) neither chamber during normal sinus rhythm, with normal atrioventricular conduction within the programmed rate limits. The DDD pacemaker also automatically converts as needed to any of these modes of pacing on a beat-to-beat basis determined by the patient's native rhythm.

The implantation of a DDD pacemaker, however, appears to be the equivalent of placing an accessory pathway in the cardiac conduction system, and there have been reports of pacemaker-mediated reentry tachycardias started by a retrograde P-wave following a premature ventricular contraction.

Necrotizing Balanitis due to Herpes Simples Type 1. Robert D. Powers, MD, Michael F. Fein, MD, and Frederick G. Hayden, MD, *Charlottesville.*

A previously healthy, 26-year-old heterosexual man experienced necrotic penile ulcerations with fever, rigors, and difficulty voiding. Urethral instrumentation and treatment with antibiotics were associated with progression of the disease. Type 1 herpes simplex virus was isolated from the penile ulcerations and pustular lesions in the groin and antecubital fossae. Recovery was complicated by a urethral stricture requiring surgical treatment. Herpes simplex type 1 must be considered in the differential diagnosis of severe penile ulcerations; isolation of herpesvirus from such lesions may obviate further diagnostic and therapeutic measures that are unnecessary and, in some cases, counterproductive. JAMA 248:215–216, 1982

Cold Urticaria with Infectious Mononucleosis: Case Report

Robert H. Anderson, MD,
Alexandria, Virginia

Case Report

A 15-year-old white male was seen on 3-9-82 complaining of urticaria of his hands and feet that had been intermittent for two days. He had white, raised papular lesions that itched, and his hands were also swollen. This had started when he was fishing without gloves. He thought it was due to the cold weather but he had never had anything like it before.

He had a temperature of 100°F but no vomiting or diarrhea. Examination showed an injected throat with enlarged, tender, anterior cervical glands. His hands and feet were normal at that time. His chest was clear. Ears were normal, as were the heart and abdomen. No splenic enlargement was noted. A throat culture was done (eventually negative for streptococci), and he was put on penicillin V by mouth.

Diphenhydramine hydrochloride (Benadryl®) was used for the urticaria. The urticaria improved, but there was a question as to whether the improvement was due to the Benadryl or the fact that he had stayed out of the cold weather.

On March 12 he returned with a history of fever and occasional urticaria of his hands. Examination showed enlargement of the anterior cervical lymph glands and of a right lateral cervical gland extending from below the ear to the back of his neck. It was not red but was tender. No urticaria was noted. Urinalysis was normal. WBC was 10,500, with 21% segs, 72% lymphs, 4% monos, 2% eosinophils and 1% basophils. There were 15 atypical lymphocytes/100 WBC. RBC was 5.03 and hemoglobin 14.9 gms. The test for mononucleosis was positive.

The patient gradually recovered over the next two weeks. The fever subsided, and the cervical lymph glands went down gradually. He had some fatigue and malaise that left in about two weeks. He has done well since.

Discussion

Cold-induced urticaria in infectious mononucleosis was first described by Tyson and Dzarny[1] in 1981 in a 24-year-old man who developed generalized urticaria and vertigo as well as upper airway obstruction and wheezing after a swim in the ocean and a run on the beach. This patient's serum showed the presence of cryoglobulins. The cold-induced urticaria was confirmed by the ice-cube test. The Paul Bunnell test for heterophile antibody was positive, as was a fluorescent antibody test for antibody against Epstein-Barr virus.

The second case in the literature was also reported in 1981.[2] This was a 19-year-old girl with glandular fever, cold urticaria and leg ulceration. She had cutis marmorata of the thighs and lower legs with some large bullae of the calves. Her urticarial wheals occured after exposure to cold and lasted about five weeks. Apparently cold agglutinins occur in about 50% of cases of infectious mononucleosis, according to Worledge and Dacie,[2] but rarely is cold urticaria seen.

A third case was reported in 1982 by Lemanske and Bush[3] in a 17-year-old boy with cold urticaria and tonsillitis with a positive culture for beta-hemolytic streptococci, nongroup A. A mononucleosis screen was positive, as was the test for qualitative cryofibrinogens in the serum. The patient had pruritic wheals on the body when exposed to cold. He was treated with oral hydroxyzine hydrochloride.

Urticaria may be seen with certain viral infections, such as hepatitis B and infectious mononucleosis. Cold-induced urticaria may occur as an inherited or acquired form[7]; the latter is more common. On exposure to cold or cold objects the patient shows a pruritic urticarial rash that may become angioedema. There may be syncope, headache or wheezing. If the whole body is cooled, as in the first

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case (swimming), there may be hypotension and potentially lethal collapse. Therefore, it is important to ask every patient with infectious mononucleosis if there have been signs of cold urticaria.

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Cure of Isolated Pulmonary Mucormycosis: Case Report

Gary K. Griffin, MD, A. Robin Ellis, MD, and Taik C. Kim, MD,
Charlottesville, Virginia

Case Report

A 64-year-old black male was admitted to the University of Virginia Hospital with an eight-year history of poorly controlled nonketotic diabetes mellitus complaining of fever, dyspnea, pleuritic chest pain and a 20-pound weight loss. Temperature on admission was 38°C. Chest films revealed a right lower lobe pneumonia. The patient received a 14-day course of high dose intravenous ampicillin. Fever and pleuritic chest pain resolved promptly. Blood cultures were negative. A single contaminated sputum culture grew *Klebsiella pneumoniae*. Hyperglycemia was controlled with subcutaneous insulin. The patient was discharged clinically improved although his chest roentgenogram continued to show residual right lower lobe infiltrate.

Three weeks later his dyspnea and pleuritic chest pain recurred with a further ten-pound weight loss. The infiltrate on chest film was unchanged following a two-week course of oral cephoradine although

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the patient reported moderate improvement in his respiratory symptoms. He was subsequently readmitted to the University of Virginia Blue Ridge Hospital Division for further evaluation of his recurrent dyspnea.

Physical exam revealed a thin black male, moderately short of breath but without cough. Temperature was 36^{5}C. Head and neck exam was unremarkable. Lung exam revealed dullness and decreased breath sounds over the right posterolateral hemithorax. The hematocrit was 34% and the white count was 11,500/mm^3 with a mild left shift. Blood sugar was 259 mg/dl. Admission chest roentgenogram revealed a patchy right lower lobe infiltrate (Fig. 1). Multiple sputum samples for bacteria, fungi, tuberculosis and malignant cells were nondiagnostic. A bronchoscopy was normal. The patient was placed on high-dose aqueous penicillin G and improved clinically. Posteroanterior and lateral chest tomograms demonstrated a triangular pleural-based density in the lateral basal segment without bronchial obstruction.

Needle aspiration biopsy was performed. Cytology preparations demonstrated broad, right-angle branching, nonseptate hyphae consistent with *Mucor* (Fig. 2). Fungal stains and cultures were negative. Repeat bronchoscopic exam with transbronchial biopsy showed no evidence of fungal infection. Open biopsy of the lung revealed a large fibrotic abscess cavity which was drained of copious amounts of yellow purulent debris. Fungal cultures were again negative. Pathologic evaluation of tissue from the cavity wall demonstrated vascular invasion by hyphae with necrosis. Intravenous amphotencin B was begun and continued to a total dose of 2 grams. The chest film revealed no residual infiltrate.

At four months followup the patient was doing well.

Fig. 1. Admission X-ray shows patchy right lower lobe infiltrate.

Fig. 2. Biopsy specimen shows broad, right-angle-branching, nonseptate hyphae consistent with *Mucor*. (×300)

Discussion

Fungi of the order Mucorales, genera *Rhizopus*, *Absidia* and *Mucor*, are considered saprophytic and generally nonpathogenic in humans, but in disease states leading to immunosuppression these fungi may be the cause of serious and fatal infections. The most common predisposing conditions are hematogenous malignancies (i.e., leukemia and lymphoma) and diabetes; pulmonary disease is seen more frequently with hematogenous malignancies. Diabetes is associated with rhinocerebral mucormycosis. Although ketosis or acidosis was once thought to be a prerequisite for infection in diabetes, mucormycosis has been diagnosed in nonketo-

tic diabetes as well. Isolated pulmonary disease is thought to be due to sport inhalation.[1] In immunocompromised hosts, the fungi have a predilection for invasion of pulmonary arteries. Intimal interruption leads to vascular thrombosis, infarction, consolidation and abscess formation.[2] Even well-localized, isolated pulmonary disease may be fatal due to massive hemoptysis.

A review of the literature reveals about 70 reported cases of isolated pulmonary infection due to *Mucor*. Fever and progressive pulmonary infiltrates in the face of antimicrobial therapy are typical, and concomitant bacterial, viral or other fungal infections may occur.[3] Radiographic presentation includes patchy infiltrates, bronchopneumonia, diffuse infiltrates, cavity formation, nodules, mass lesions or fungus balls.[4] Only about 15% of cases in the literature have yielded positive cultures. Transbronchial biopsy by flexible bronchoscopy may demonstrate parenchymal invasion by the fungi. Closed needle aspiration biopsy is gaining popularity as a relatively safe and reliable method for obtaining tissue samples. Cytology preparations may be prepared and interpreted rapidly and accurately.[5] Of the reported cures, identification of the infection was made by culture in two cases, bronchoscopy in five cases, aspiration needle biopsy in three cases and surgery in seven cases. In the majority of additional reported cases diagnosis was made at autopsy.[2,3]

Preferred treatment for localized pulmonary mucormycosis consists of lobectomy or wedge resection combined with amphotericin B. Systemic amphotericin B alone is reserved for cases of disseminated infection or when surgery is not feasible.[6-8] Cure was obtained by resection alone in five cases.[3] Two others had surgery combined with some other forms of therapy but were essentially surgical cures.[3] In seven other cases, including the one presented here, surgery was combined with either preoperative or postoperative amphotericin although some patients received minimal doses.[5,13-15]

Patients with hematologic malignancies comprise about three-quarters of total reported cases,[3] while they represent only 18% (three cases) of cures. Diabetics comprise about 25% of all cases reviewed[11] and about 60% (ten cases) of total cures. The diabetic patient generally is better able to undergo surgical resection, which is important to successful management of pulmonary mucormycosis.

The authors thank Mr. Gregory Fortier and Dr. Loulie M. Fisher for their clinical assistance.

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VIRGINIA MEDICAL

EDITORIAL

A Neat Trick

LIVER BORDERS can be devilish to find, especially in people who are obese. Methods usually used in this endeavor include palpation, hard percussion and light percussion. Very light percussion as described by Castell et al[1] is a fairly successful technique.

If, after using these approaches one is still unable to locate the lower edge of the liver, one might try the following trick: Apply the diaphragm portion of the stethoscope to a remote area of the abdomen—left lower quadrant is satisfactory—and listen while lightly tapping with a finger the right side of the abdomen, starting in the lower quadrant and advancing cephalad. The lower border will be appreciated when a change in tone occurs, especially a flat tone. This maneuver should be repeated with the stethoscope applied to various parts of the abdomen to confirm the location.

The upper border may be discovered in the same fashion, with the stethoscope applied high on the chest wall, the finger tapping from above downward. The upper border is usually found in the fourth intercostal space; if it is in the fifth or lower, then the diaphragm is displaced downward, as in emphysema.

According to Castell and also to Sapira and Williamson,[2] the span of the liver varies normally with height, body mass and age—the span increasing with age. It is also longer in males. The span in most middle-aged adults in the mid-clavicular line is about 14 centimeters. Livers greater than 15 centimeters are probably enlarged. In addition to the size, it is well to keep in mind that diseases of the liver also affect its consistency, causing it to be firm.

These maneuvers are performed while the patient is supine and breathing quietly. Most reproducible results are obtained while the patient is at end-tidal volume, especially if one is measuring the distance between the costal margin and the lower edge of the liver.

Finally, one may also evaluate the spleen in this fashion, recalling that the spleen generally follows the course of the tenth rib, with the anterior aspect of the spleen extending to the mid-axillary line. Slight enlargement of the spleen may be detected with the stethoscope applied to the abdominal wall and percussing from the left upper quadrant towards the anterior axillary line in the tenth interspace. A spleen that has become palpable is, after all, a fairly large spleen; this method detects splenic enlargement before it reaches that size.

ROBERT A. ABERNATHY, MD

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VIRGINIA MEDICAL OBITUARY

C. F. Johnston, Jr., MD

Dr. Clarence Frederick Johnston, Jr., longtime Abingdon surgeon, died June 4 at Johnston Memorial Hospital, Abingdon. He was 72 years old and had retired from practice in 1976.

A Baltimore native, Dr. Johnston was graduated from Johns Hopkins University and received his medical degree in 1937 from the University of Maryland School of Medicine. He trained at Maryland General Hospital, Baltimore, and the Mayo Clinic. During World War II Dr. Johnston served the US Army as a colonel and chief of surgery of the 133rd Evacuation Hospital in Europe. He practiced in Baltimore and Grundy, Virginia, before moving to Abingdon in 1948.

Dr. Johnston was a member of the Southwestern Virginia Medical Society and had belonged to The Medical Society of Virginia since 1947. He belonged also to the American College of Surgeons and American Society of Abdominal Surgeons.

W. W. Waddell, Jr., MD

Dr. William Wirt Waddell, Jr., a charter member and past president of the Virginia Pediatric Society, died on June 7 in Bristol, Tennessee, at the age of 89.

A native of Charlottesville, Dr. Waddell received his doctorate in medicine from the University of Virginia School of Medicine in 1918. Joining his alma mater's Department of Pediatrics in 1924, he served as its chairman for 19 years and on his retirement in 1965 was named professor emeritus. Dr. Waddell is said to have been the first to use Vitamin K for the treatment of prothrombin deficiency and hemorrhagic disease in the newborn, and he pioneered in the administration of pertussis vaccine to infants. These and other innovative procedures and studies he recorded for the literature with a total of 34 articles. In cooperation with the Charlottesville and Albemarle County health departments, Dr. Waddell established a well-child clinic program and was its attending physician for 40 years.

His membership in The Medical Society of Virginia spanned half a century and he belonged also to the Albemarle County Medical Society, American Academy of Pediatrics and American Medical Association.

R. Earle Glendy, MD

Dr. Robert Earle Glendy, chairman emeritus of the Department of Cardiology at Roanoke Memorial Hospital, died at that hospital on June 26. He was 81 years old and had retired in 1977.

A Columbus, Ohio, native, Dr. Glendy was graduated from Virginia Military Institute in 1925 and earned his medical degree from the University of Virginia in 1931. He received training at Peter B. Brigham and Massachusettes General Hospitals, and from 1936 to 1941 taught at Harvard Medical School. He joined the Army Medical Corps in 1941 and at the end of World War II settled in Roanoke.

Dr. Glendy had been president of the Roanoke Academy of Medicine and of the Virginia Heart Association. A member of the American College of Physicians and the American Society of Internal Medicine, his membership in The Medical Society of Virginia spanned 37 years.

Thomas H. Chun, MD

Dr. Thomas HwaYoung Chun, chairman of the Department of Anesthesiology at St. Mary's Hospital, Richmond, and clinical professor of anesthesiology at the Medical College of Virginia, died at home on July 6 after a long illness. He was 56 years old.

Dr. Chun was a native of Hwang Hae Do, Korea, and received his medical degree in 1952 from the Medical College of Seoul National University. He came to the United States in 1953 and worked in several hospitals in the United States and Canada before moving to Richmond in 1964. On June 26, 1983, Dr. Chun received the South Korean government's highest civilian award for service to the Korean people and that nation; named Mu Kung Wha, after the Korean national flower, the gold medal has been given to only three other persons.

Dr. Chun was a past president of the Korean Medical Association in North America and be-

longed also to the American College of Anesthesiologists and the International Anesthesia Research Society. He came to membership in The Medical Society of Virginia through the Richmond Academy of Medicine.

C. W. Hickam, Sr., MD

Dr. Cecil William Hickam, Sr., family practitioner in Pulaski for 45 years, died at the age of 78 on June 8, in Pulaski Community Hospital. He had retired from practice last year.

A native of Lee County, Dr. Hickam was graduated from Washington and Lee University in 1925 and from the University of Pennsylvania School of Medicine, Philadelphia, in 1931. Following training in Cincinnati, Ohio, Dr. Hickam returned to Virginia in 1937 and established his practice in Pulaski. He was one of the founders of the Pulaski Clinic and had been a member of the Southwestern Virginia Medical Society and The Medical Society of Virginia since 1940. He was also a member of the American Academy of Family Physicians.

A son, Dr. G. Lindsay Hickam practices medicine in Roanoke.

Memoir of C. B. Trower 1912–1982

By Charles E. Davis, Jr., MD, Jeremiah Klotz, MD, and Charles VanHorn, MD

Dr. Clarence B. Trower, who practiced surgery in Norfolk for 25 years, died on September 10, 1982. Except for the time he spent at the University of Virginia obtaining his BA and MD degrees, he lived always in his native Norfolk. His postgraduate surgical education was obtained over a five-year period at Norfolk General Hospital, and he was an active member of Norfolk General's staff throughout his practice.

Dr. Trower belonged to a number of civic, fraternal, and professional organizations and clubs and held high office in many of them. Continuously for nine years he represented the Second District on the State Board of Medicine; this service culminated in his election to the presidency of this important and prestigious body. His conscientious, thorough, and compassionate approach to the multiple problems, often vexing and poignant, of fellow physicians appearing before the Board, was exemplary and deeply appreciated. His resignation in 1980 because of ill health was a great loss.

He served for many years as the recording secretary of the Department of Surgery at Norfolk General Hospital. His detailed records of the actions of that body, often quite complex, remain models of accuracy, thoroughness and diplomacy. He was a valued member of the Cosmopolitan Club, and in its weekly bulletin of September 16th, 1982, that organization emphasized his friendship and many contributions in a magnificent tribute. He was a dedicated father to his family and a tower of strength to them. His sister, Ann, her husband, and three children survive him.

Clancy Trower loved people. His gregarious personality and warm human qualities provided him with a host of friends, not only in his profession but throughout the entire community. Very rarely has a physician enjoyed the close friendships of so many prominent non-medical persons. He frequently entertained in his lovely home, an activity he thoroughly enjoyed until shortly before his death.

Several years prior to his retirement and subsequent death he was strickened with a progressively debilitating and devastating illness. His associates, patients and friends sadly and helplessly watched the inexorable ravages of this disease and its effects on him. All were proud of the magnificent but futile battle he waged against it. His last few years were indeed a lesson in courage.

Hubert B. Holsinger, MD

Dr. Hubert B. Holsinger, for 37 years a surgeon in Farmville, died June 21 at the age of 79. He had retired from practice in 1969.

A native of Rockingham County, Dr. Holsinger was graduated from the University of Virginia School of Medicine in 1928 and trained at the University of Virginia and Yale University. He served during World War II with the Army Medical Corps in Europe.

A member of The Medical Society of Virginia for more than 50 years, Dr. Holsinger was also a member of the Southside Virginia Medical Society, Southeastern Surgical Congress, American College of Surgeons and the American Society of Abdominal Surgeons.

WHO'S WHO

A new Cancer Center has opened at the Medical College of Virginia/Virginia Commonwealth University, with **Dr. Walter Lawrence, Jr.,** as director and a staff of 100 clinical specialists and scientists. The dedication ceremonies, with Dr. Lawrence presiding, drew Gov. Charles S. Robb, who was introduced by **Dr. Edmund F. Ackell,** VCU president.

At the time of the opening ceremonies only two floors of the new building had been completed; these are devoted in large part to expanded facilities for radiation therapy, which draws as many as 200 persons in a single day to the MCV complex in downtown Richmond. Yet to come are the two floors for research laboratories and a new Joint Cancer Clinic, where outpatients will be treated. The completed structure is expected to cost $8.5 million; it is being financed by state allocations, bond issues and private donations.

Dr. Lawrence is chairman of surgical oncology at MCV. Other Medical Society of Virginia members who have leading roles in the new Center's services are **Dr. I. David Goldman, Jr.,** chairman, medical oncology; **Dr. Tapan A. Hazra,** chairman, radiation therapy and oncology; **Dr. Dean R. Goplerud,** director, gynecologic oncology; **Dr. G. Watson James III,** chairman, hematology; **Dr. Susan J. Mellette,** director, cancer rehabilitation and continuing care; and **Dr. J. Shelton Horsley III,** director, Joint Cancer Clinic.

New Governor for Virginia of the American College of Physicians is **Dr. James P. Baker,** acting dean at Eastern Virginia Medical School, Norfolk. He will hold the post for four years.

Dr. David M. Pariser, Norfolk, **Dr. James Ellis Dill,** Roanoke, and **Dr. Daniel M. Goodenberger,** Alexandria, are newly elected ACP fellows.

Named Norfolk's First Citizen of 1982 by the Cosmopolitan Club of Norfolk was **Dr. Robert L. Payne, Jr.,** retired surgeon who was honored for "a lifetime of medical, civic and philanthropic accomplishments," including a key role in the creation of Eastern Virginia Medical School.

A Norfolk native, Dr. Payne was an Army surgeon during World War II, then joined his father, also a surgeon, in practice. He helped found the Virginia Surgical Society and the American Society for Surgery of the Hand and has been president of the Southern Society of Clinical Surgeons. For four years chairman of the Eastern Virginia Medical Authority, he is today the Authority's secretary and heads the development committee of the Eastern Virginia Medical Foundation.

The medical staff of Petersburg General Hospital has elected **Dr. W. Benjamin McIlwaine III,** general practitioner, as its president.

Dr. Gustavus V. Jackson, Jr., of Richmond, medical director of C&P in Virginia, has been elected president of the Virginia Chapter, American Occupational Medical Association.

Elected to not one but two offices at a recent meeting of the Society of Cardiovascular and Interventional Radiology was **Dr. Charles J. Tegtmeyer,** Charlottesville, who was named both president-elect and secretary-treasurer. Professor of radiology and associate professor of anatomy at the University of Virginia, Dr. Tegtmeyer is widely known for his landmark work with percutaneous transluminal angioplasty.

Dr. John A. Wente is the new president of the Bedford County Medical Society and **Dr. Eileen T. Jennings** has been reelected secretary. Both practice in Bedford.

New president of the Alexandria Hospital medical staff is **Dr. Michael H. Friedman,** radiologist.

Dr. George L. Fischer, Clifton Forge, is the new president of the Virginia Society of Internal Medicine. Elected to serve with him were **Dr. William C. Branscome,** Staunton, president elect; **Dr. John W. Knarr,** Pulaski, vice president; and **Dr. W. Wayne Key, Jr.,** Richmond, secretary-treasurer.

Dr. Walter C. Elliott, widely known in Southwest Virginia both as a physician and as a former member of Virginia's House of Delegates, marked his 80th birthday early this year by announcing his retirement. He had been practicing in Lebanon, Russell County, for 50 years.

Born in Campbell County, Dr. Elliott was educated at the University of Richmond and the Medical

College of Virginia (Class of 1928). He first practiced in a coal camp but soon settled in Lebanon, where, to serve his general practice, he took over a 12-bed hospital on the second floor of a commercial building. When that facility flourished, he built a new 25-bed hospital, naming it Lebanon General Hospital; it now has over 100 beds, is owned by a corporation, and is called the Russell County Medical Center.

Dr. Elliott's service as a delegate to the General Assembly from 1952 to 1964 was described in the January 1981 issue of VIRGINIA MEDICAL in an article titled "Serving a Locum Tenens in Virginia's Statehouse."

In the 1940s, **Dr. James W. Elliott** joined his brother as a partner; he continues the Lebanon practice.

Dr. Lockhart B. McGuire, cardiologist in the University of Virginia's Department of Internal Medicine, has been named the first Julian Ruffin Beckwith Professor of Medicine by the university's board of visitors. A graduate of the University of Virginia School of Medicine, Dr. McGuire joined its faculty in 1964. Dr. Beckwith, who died last year, headed the university's division of cardiology from 1962 to 1977.

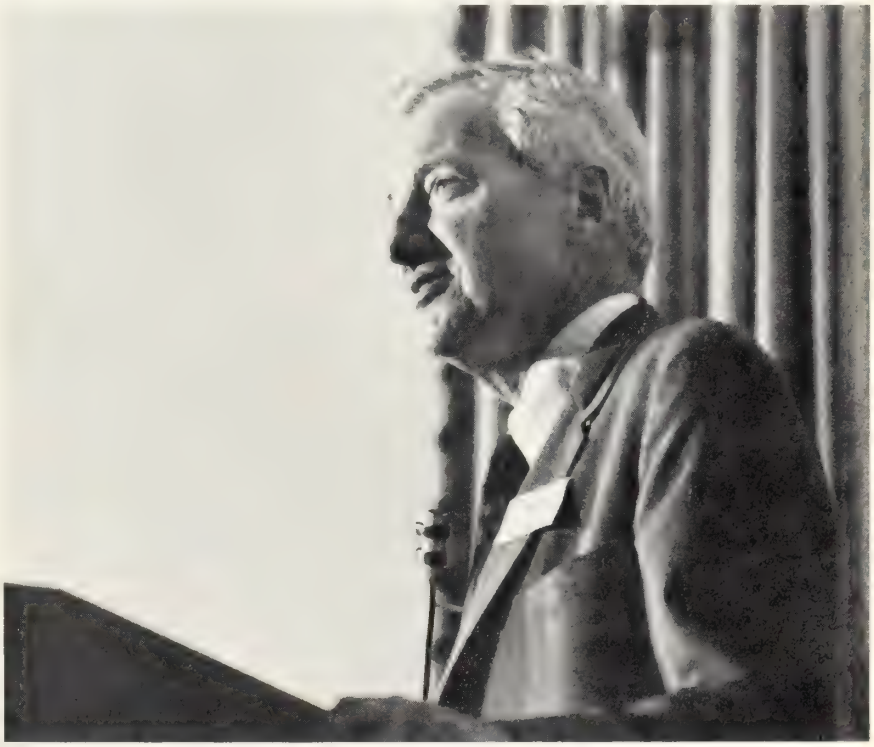
New president of the Neuropsychiatric Society of Virginia is **Dr. Wesley B. Carter,** Richmond, who is a past president of both the Richmond Medical Society and the Virginia Council on Child Psychiatry.

Recipient of the 1983 Distinguished Young Physician Award of the University of Missouri Medical Alumni Association was **Dr. John B. McCraw,** professor of plastic surgery at Eastern Virginia Medical School. Dr. McCraw received both his BA and MD degrees from the University of Missouri.

New officers of the Rockbridge County Medical Society include these two Medical Society of Virginia members: **Dr. William E. Weddle,** Buena Vista, president, and **Dr. Sanford B. Radom,** Lexington, secretary-treasurer.

Dr. Lawrence A. Bernet, Jr., Norfolk psychiatrist, has been installed as the president of the Norfolk Academy of Medicine. Serving with him for 1983-1984 are **Dr. Frank W. Gwathmey,** president elect; **Dr. George H. M. Rector,** vice president; **Dr. Jack D. Mahan,** secretary; and **Dr. Mark T. Schreiber,** treasurer.

To **Dr. Melissa A. Warfield**, Norfolk pediatrician, went the 1983 Woman of the Year title conferred annually by a coalition of businesses in the Tidewater area. Dr. Warfield is director of the division of hematology and oncology at Children's Hospital of the King's Daughters in Norfolk and professor of pediatrics at Eastern Virginia Medical School. She is a graduate of Women's Medical College of Pennsylvania and interned at Norfolk General Hospital.



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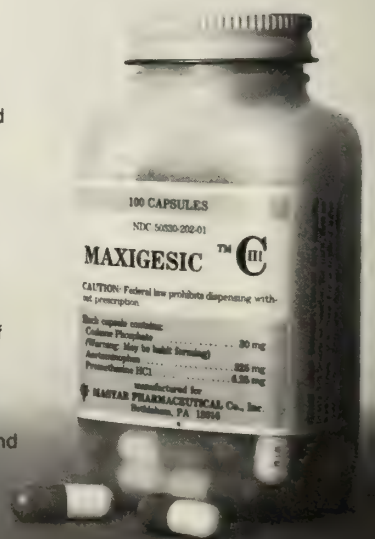

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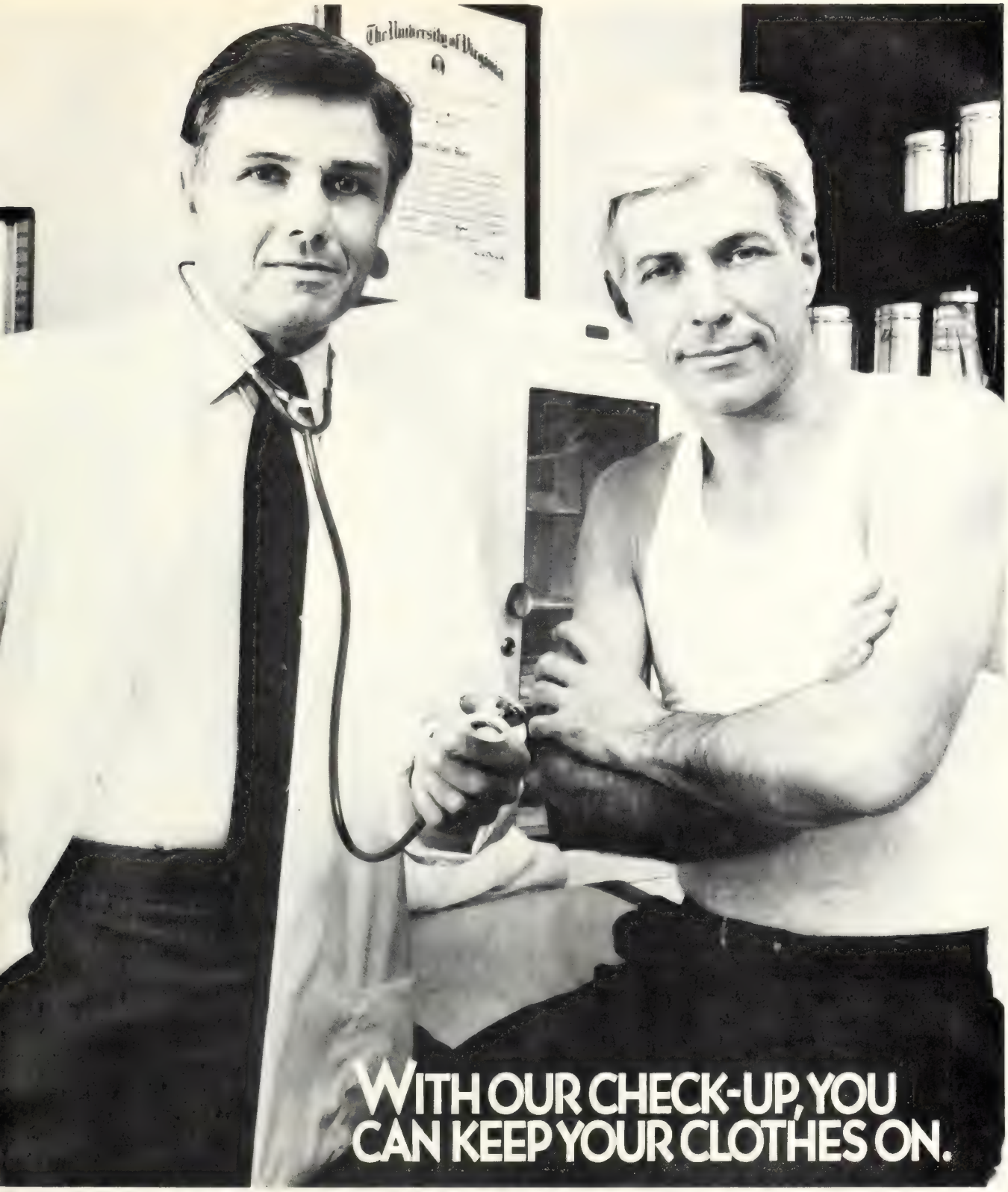

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WARNINGS: Ru-Tuss Tablets may cause drowsiness. Patients should be warned of possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers.

PRECAUTIONS: Ru-Tuss Tablets contain belladonna alkaloids, and must be administered with care to those patients with urinary bladder neck obstruction. Caution should be exercised when Ru-Tuss Tablets are given to patients with hypertension, cardiac or peripheral vascular disease or hyperthyroidism. Patients should avoid driving a motor vehicle or operating dangerous machinery (See WARNINGS:).

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References: 1. Kales A *et al: J Clin Pharmacol 17*:207-213, Apr 1977 and data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Kales A: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **3.** Zimmerman AM: *Curr Ther Res 13*:18-22, Jan 1971. **4.** Kales A *et al: JAMA 241*:1692-1695, Apr 20, 1979. **5.** Kales A, Scharf MB, Kales JD: *Science 201*:1039-1041, Sep 15, 1978. **6.** Kales A *et al: Clin Pharmacol Ther 19*:576-583, May 1976. **7.** Kales A, Kales JD: *Pharmacol Physicians 4*:1-6, Sep 1970. **8.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **9.** Dement WC *et al: Behav Med 5*:25-31, Oct 1978. **10.** Vogel GW: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **11.** Karacan I, Williams RL, Smith JR: The sleep laboratory in the investigation of sleep and sleep disturbances. Scientific exhibit at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971. **12.** Pollak CP, McGregor PA, Weitzman ED: The effects of flurazepam on daytime sleep after acute sleep-wake cycle reversal. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, June 30-July 4, 1975. **13.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ.

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Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

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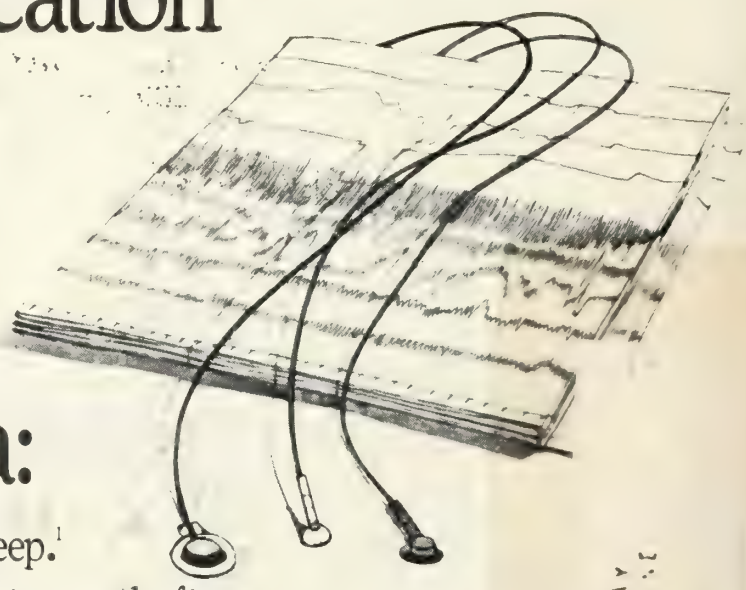

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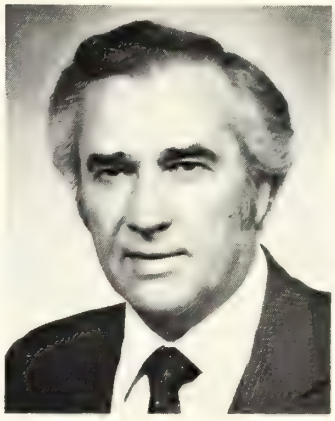

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October 1983, Volume 110, Number 10

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On the cover **Dr. John A. Martin, Roanoke, speaks before the House of Delegates at the 1982 Annual Meeting**

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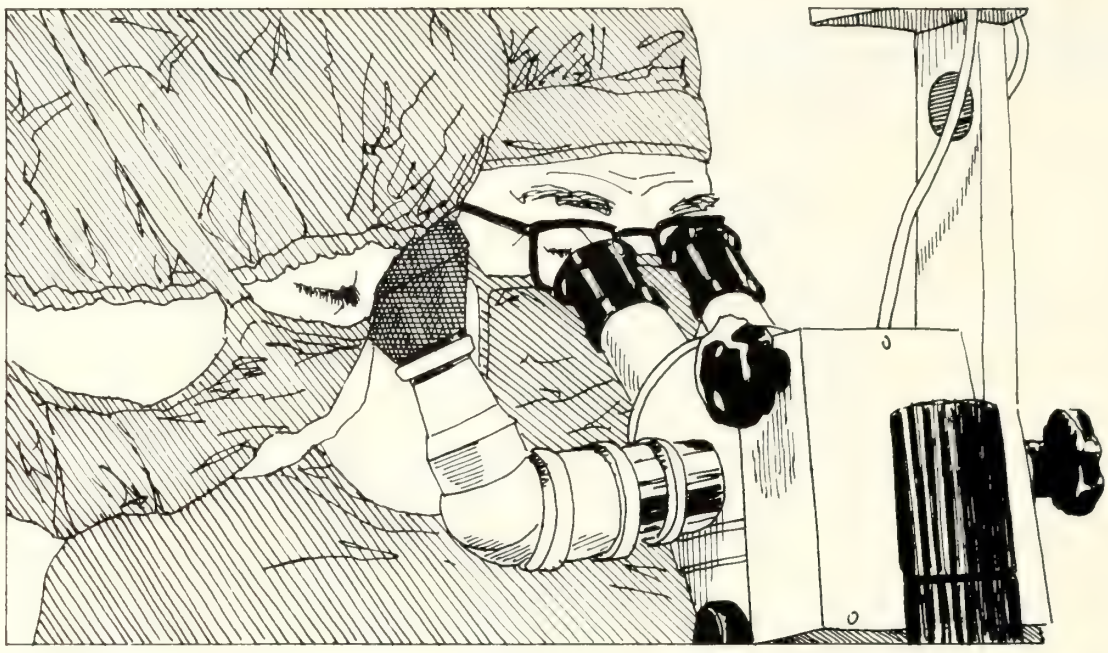

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LETTERS

Applause for editorial seeking Virginia Council support

Many letters were received in response to the Editor's recent plea for stronger physician support of the Virginia Council on Health and Medical Care. These are some of them.

I would like to commend you on the excellent editorial concerning the Virginia Council on Health and Medical Care which appeared in the July issue of VIRGINIA MEDICAL.[1]

An important point was made in your editorial which needs reemphasizing. Although many physicians in the state have been helped by the Council in finding an appropriate place to practice, a disappointingly small percentage (6%) have contributed to the work of the council. Physicians frequently complain about governmental interference, both on the federal and state level, yet when a completely privately funded organization such as the Virginia Council on Health and Medical Care, unfettered by governmental strings, asks for our support to enable it to carry out its important work, we are slow to respond. We can all do a better job in supporting this important organization and, as you said in your editorial, let's do it now.

Daniel N. Mohler, MD

University of Virginia School of Medicine
Charlottesville VA 22908

1. Kendig ELjr: Does your generosity need exercising? Start here. Va Med 110:451, 1983

I want to compliment the editorial in the July 1983 issue of VIRGINIA MEDICAL on the Virginia Council on Health and Medical Care. Throughout all of the years in which there was an acute shortage of physicians the Virginia Council on Health and Medical Care represented conscientious efforts by physicians to try to place needed medical care in appropriate areas, and we can point with pride to its successes.

Time changes and new challenges appear but the Virginia Council on Health and Medical Care represents the finest voluntary effort to solve health care problems in the state and deserves every physician's financial support.

Charles E. Horton, MD

400 West Brambleton Avenue
Norfolk VA 23510

I want to congratulate you on the editorial in July 1983 issue recommending support for the Virginia Council on Health and Medical Care.

Mr. Fisher and his organization have been extremely helpful to us at the Manassas Medical Center, in helping us obtain physicians for our organization and for the area of Prince William County during its growing years. This privately supported organization deserves all the support it can get and especially from the physicians in Virginia. I agree with the editorial wholeheartedly.

Robert A. W. Latimer, MD

9202 Centreville Road
Manassas VA 22110

Let me second the July editorial about our physicians contributing more generously to the Virginia Council on Health and Medical Care.

As one who was placed on the Eastern Shore of Virginia by the Council 25 years ago, I remain indebted to that organization. Most impressed with their activities, I know dollars contributed are wisely spent!

Please, physicians of Virginia respond to the need for further funding; it may avoid governmental interference.

William F. Bernart, MD

Internal Medicine Group
Nassawadox VA 23413

I read with interest the editorial in VIRGINIA MEDICAL's July issue describing the many worthwhile activities of the Virginia Council on Health and Medical Care.

The services offered certainly are valuable to physicians as well as to the allied health professions. This organization deservedly merits the support of all physicians in Virginia, and I urge them to respond.

William T. Wilkins, MD

A. H. Robins Company
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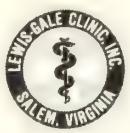

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MEETINGS ABOUT MEDICINE

October 21
Fall Meeting: Virginia Occupational Medical Association, *Roanoke*. 5 credit hrs. Gus Jackson, MD, 5801 Bremo Rd., Richmond VA 23226, (804) 281-8505.

October 27-29
Anesthesiology Review Seminar (American Academy of Clinical Anesthesiologists/East Tennessee State University), *Gatlinburg, Tennessee*. Donald G. Catron, MD, PO Box 11691, Knoxville TN 37919, (615) 588-6279.

October 28-29
Advances in Echocardiography (Virginia Heart Institute), *Richmond*. Jane B. Baird, 205 N. Hamilton St., Richmond VA 23221, (804) 359-9265.

October 31-November 2
Mechanisms of Neurotoxicity (Johns Hopkins University), *Baltimore*. Jacqueline Corn, Continuing Education, 615 N. Wolfe St., Baltimore MD 21205, (301) 955-2609.

November 2
Management of Stress (East Tennessee State University), *Bristol, Tennessee*. Carol Blevins, Bristol Family Practice Clinic, 100 Arapahoe Dr., Bristol TN 37620, (615) 968-5212.

November 2-4
Advanced Obstetrics and Gynecology (Johns Hopkins University), *Baltimore*. 24 Cat. I credits. Fee: $300. Joan Batt, Dept. of Radiology, Johns Hopkins Hospital, Baltimore MD 21205, (301) 955-8450.

November 3-4
Current Topics in Urology (University of North Carolina), *Chapel Hill*. Floyd A. Fried, MD, Div. of Urology, UNC, Chapel Hill NC 27514, (919) 966-2571.

November 3-5
Antenatal Diagnosis and Treatment of Fetal Disease (Medical College of Virginia), *Williamsburg*. 14½ Cat. I credits. Fee: $275 for MDs; $175 for residents, interns. Sheri Rosner, Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

November 4-5
Asthma (American Lung Association of Virginia, Southwest Region/East Tennessee State University), Abingdon. Anne Hall, Virginia Lung Association, 524 Cumberland, Bristol VA 24201, (703) 669-2933.

November 5-6
Reading Retreat: Recent Advances in Cardiology and Gastroenterology (Virginia Chapter of American College of Physicians), *Peaks of Otter Lodge*. 15 Cat. I credits. Fee: $100. Daniel N. Mohler, MD, University of Virginia Medical Center, Box 395, Charlottesville VA 22908, (804) 924-5118.

November 6-9
Annual Meeting: American Physicians Art Association, *Baltimore*. S. W. Parks. MD, 710 Myrtle Rd., Charleston WV 25314.

November 6-9
77th Annual Scientific Assembly: Southern Medical Association, *Baltimore*. Ms. Jeanette Stone, SMA, PO Box 2446, Birmingham AL, 35201, (205) 323-4400.

November 9
Respiratory Care Update (American Lung Association of Virginia, Richmond Region/Virginia Commonwealth University School of Nursing), *Richmond*. Shirley Lipscomb, American Lung Assoc. of Virginia, PO Box 7065, Richmond VA 23221, (804) 355-3295.

November 9
The Management of Stress (East Tennessee State University), *Bristol, Tennessee*. Carol Blevins, MSW, Bristol Family Practice Clinic, 100 Arapahoe Dr., Bristol TN 37620, (615) 968-5212.

November 11
Disaster Planning and Management (Virginia Chapter, American College of Emergency Physicians), *Williamsburg*. 7 Cat. I credits. Gwen E. Messler, PO Box 317, Highland Springs VA 23075, (804) 737-9433.

November 11-13
Update in Infectious Diseases (Johns Hopkins University), *Ocean City, Maryland*. 10 Cat. I credits. Sandy Young, Office of CME, 720 Rutland Ave., Baltimore MD, 21205, (301) 955-5880.

continued over

November 15-20
Gynecologic Endoscopy and Microsurgery (American Association of Gynecologic Laparoscopists), *Washington DC*. J. M. Phillips, MD, 11239 S. Lakewood Blvd., Downey CA 90241.

November 17
Annual Program on Cancer (Roanoke Memorial Hospital), *Roanoke*. Charles L. Crockett, Jr., MD, Roanoke Memorial Hospital, Roanoke VA 24033, (703) 981-7228.

November 18-19
Frontiers of Gynecologic Oncology (University of Maryland), *Baltimore*. Office of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

November 18-19
Progress in Pediatrics (Johns Hopkins University), *Baltimore*. 11 Cat. I credits. Fee: $80 for MDs; $40 for residents. Noreen Javornik, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-6046.

December 1-3
Comprehensive Breast Disease Symposium (Bowman Gray School of Medicine), *Winston-Salem, North Carolina*. 20 Cat. I credits. Fee $300. James F. Martin, MD, 300 S. Hawthorne Rd., Winston-Salem NC 27103, (919) 748-4505.

December 3
11th Annual Symposium on Geriatric Medicine (Johns Hopkins University), *Baltimore*. 5½ Cat. I credits. Fee: $40. Susan Bavaro, Office of CME, 720 Rutland Ave., Baltimore MD 21205 (301) 955-6046.

December 8-10
International Helen B. Taussig Symposium in Pediatric Cardiology (Johns Hopkins University/American Heart Association), *Baltimore*. Cat. I credits. Fee: $300. Carlita M. Kearney, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

December 8-10
Pediatric Imaging Seminar (Bowman Gray School of Medicine), *Winston-Salem, North Carolina*. 20 Cat. I credits. Thomas E. Sumner, MD, 300 S. Hawthorne Rd., Winston-Salem NC 27103, (919) 748-4505.

December 10
Anxiety and Depression in Office Patients (Eastern State Hospital/Taylor Manor Hospital), *Williamsburg*. 6 Cat. I credits. Fee: $50. Mary Ann Ayd, PO Box 16315, Baltimore MD 21210, (301) 435-6562.

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Yet another benefit Valium affords is flexibility. Available in 2-mg, 5-mg and 10-mg scored tablets, Valium enables you to titrate dosage to individual patient needs. For the geriatric patient, a starting dosage of 2 to 2½ mg once or twice a day is recommended. And, for patients who forget or skip medication, you can prescribe Valrelease™ (diazepam/Roche) 15-mg slow-release capsules, knowing that Valrelease will assure all the benefits of Valium 5 mg *t.i.d.* with the convenience of once-a-day dosage.

Discontinuation of Valium (or Valrelease) is typically as smooth as its start in short-term therapy. However, Valium and Valrelease should be discontinued gradually after more extended treatment. As you diminish dosage, the built-in tapering action of Valium and Valrelease will help avoid rapidly recurring anxiety symptoms and symptoms of withdrawal, and will help ease the patient's transition to independent coping when therapeutic goals have been achieved.

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For a summary of product information, please turn the page.

ROCHE®

Valium® (diazepam/Roche) Ⓝ Tablets
Valrelease™ (diazepam/Roche) Ⓝ slow-release Capsules
Injectable Valium® (diazepam/Roche) Ⓝ

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, impending or acute delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: relief of skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome. *Oral forms* may be used adjunctively in convulsive disorders, but not as sole therapy. *Injectable form* may also be used adjunctively in: status epilepticus; severe recurrent seizures; tetanus; anxiety, tension or acute stress reactions prior to endoscopic/surgical procedures; cardioversion.

The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence

***Usage in Pregnancy*: Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins, i.e., dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated)

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.

INJECTABLE Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance

INJECTABLE Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia

In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported

Dosage: Individualize for maximum beneficial effect.

ORAL *Adults*: Anxiety disorders, relief of symptoms of anxiety—Valium (diazepam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months)

INJECTABLE Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.)

For dosages in infants and children see below; have resuscitative facilities available

I.M. use: by deep injection into the muscle

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children *administer I.V. slowly*; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed

Respiratory assistance should be available

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful

In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension. Dialysis is of limited value

How Supplied:

ORAL Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100 and 500; Prescription Paks of 50, available in trays of 10; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25 and in boxes containing 10 strips of 10.

Valrelease (diazepam/Roche) slow-release capsules—15 mg (yellow and blue), bottles of 100; Prescription Paks of 30

INJECTABLE Ampuls, 2 ml, boxes of 10; Vials, 10 ml, boxes of 1; Tel-E-Ject® (disposable syringes), 2 ml, boxes of 10. Each ml contains 5 mg diazepam, compounded with 40% propylene glycol, 10% ethyl alcohol, 5% sodium benzoate and benzoic acid as buffers, and 1.5% benzyl alcohol as preservative

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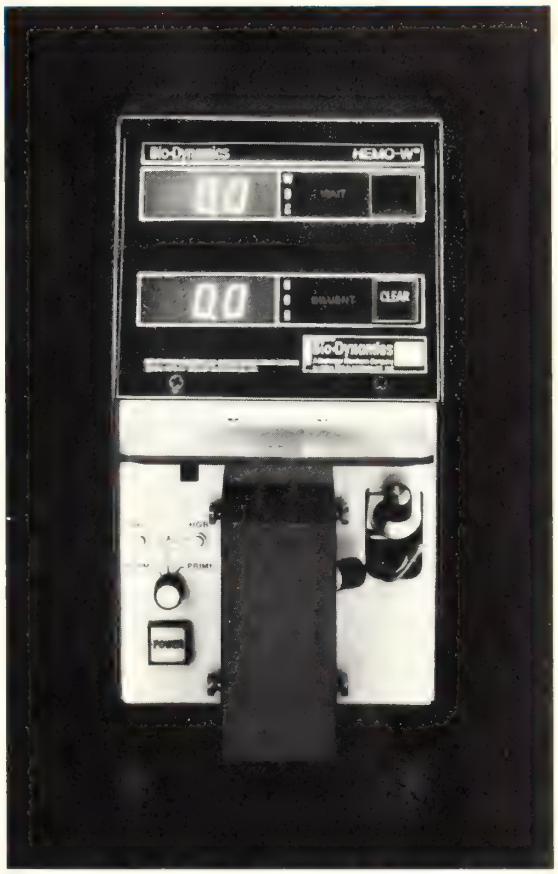

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J-9043-4 July 1981

In vitro studies demonstrate

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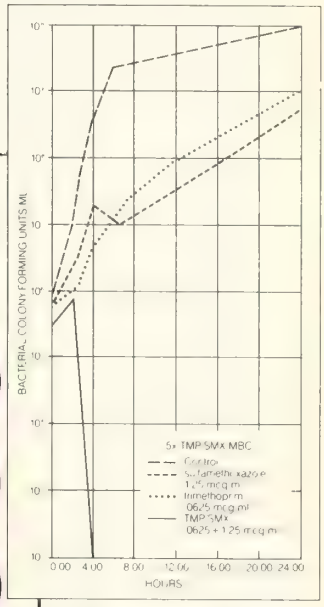
Percent of isolates of common uropathogens sensitive to BACTRIM and to other antimicrobials

	Escherichia coli	*Klebsiella pneumoniae*	*Proteus mirabilis*	*Proteus vulgaris*	*Proteus sp.*	*Enterobacter sp.*	*Enterobacter aerogenes*	*Enterobacter cloacae*
BACTRIM	**96%** 328,598	**89%** 63,551	**93%** 62,691	**84%** 4174	**91%** 4381	**88%** 1736	**96%** 7326	**92%** 11,856
ampicillin	72% 345,209	5% 65,466	85% 67,338	16% 4381	70% 4402	10% 1775	9% 7759	11% 12,091
cephalexin†	81% 343,339	85% 67,031	92% 67,027	16% 4398	80% 4365	22% 1834	13% 7571	12% 11,846
nitrofur-antoin	97% 307,785	65% 61,162	7% 59,775	13% 3848	14% 3059	67% 1707	60% 7109	67% 10,830

†Analogous to cephalothin, the primary antibiotic disc used in testing.
Source: The Bacteriologic Report, BAC-DATA Medical Information Systems, Inc., Winter Series, 1981-82.
Numbers under percentages refer to the projected number of isolates tested.

RAPID *IN VITRO* DESTRUCTION OF *E. COLI**



Kill curve kinetics of Bactrim and its individual components against *E. coli in vitro*.[1]

The bactericidal action of Bactrim has been demonstrated *in vitro* on laboratory strains of *E. coli*[1,2] and on clinical isolates of *E. coli, Klebsiella-Enterobacter, Proteus mirabilis* and *Morganella morganii*[3]—the most common causative organisms of urinary tract infections.[4] More than 100 published studies attest to the efficacy of Bactrim in recurrent urinary tract infections due to these organisms.[5] In comparative studies with other antimicrobials, Bactrim has consistently demonstrated unsurpassed efficacy during therapy.[6-11]

Resistance to Bactrim develops more slowly than to either of its components alone *in vitro*.* Among urinary tract isolates, resistance has rarely emerged in susceptible strains.[5,12] Bactrim is contraindicated in pregnancy at term, during lactation, in infants less than two months old and in documented megaloblastic anemia due to folate deficiency.

Initial episodes of uncomplicated urinary infections should be treated with a single-agent antimicrobial.

Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

b.i.d. *for recurrent urinary tract infections*

**In vitro* data do not necessarily predict clinical results.

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See next page for references and a summary of product information.

References: 1. Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 2. Kramer MJ, Mauriz YR, Robertson TL, Timmes MD: Morphological studies on the effect of subinhibitory and inhibitory doses of sulfamethoxazole-trimethoprim combination on *Escherichia coli*. Presented at the 12th International Congress of Chemotherapy, Florence, Italy, Jul 19-24, 1981. 3. Spicehandler J *et al: Rev Infect Dis* 4:562-565, Mar-Apr 1982. 4. Stamey TA: *Pathogenesis and Treatment of Urinary Tract Infections*. Baltimore, Williams & Wilkins, 1980, p. 13. 5. Ronald AR: *Clin Ther* 3:176-189, Mar 1980 6. Cooper J, Brumfitt W, Hamilton-Miller JMT: *J Antimicrob Chemother* 6:231-239, 1980. 7. Gower PE, Tasker PRW: *Br Med J* 1:684-686, Mar 20, 1976. 8. Cosgrove MD, Morrow JW: *J Urol* 111:670-672, May 1974. 9. Iravani A *et al: Antimicrob Agents Chemother* 19:598-604, Apr 1981. 10. Schaeffer AJ, Flynn S, Jones J: *J Urol* 125:825-827, Jun 1981. 11. Rous SN: *J Urol* 125:228-229, Feb 1981. 12. BAC-DATA Medical Information Systems, Inc., Bacteriologic Reports, Winter Series, 1976-82

Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii*. It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients. *Pregnancy:* Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies

Dosage: *Not recommended for infants less than two months of age.*

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 20. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per tea spoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint)

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The Medical Society of Virginia
PROCEEDINGS

Annual Report of the Executive Vice President

There is no doubt that The Medical Society of Virginia had an absolutely super year. We participated in the International Summit Conference of Industrialized Nations in Williamsburg. Our program to help impaired physicians began to function on July 11. Initial steps were taken to establish a Medical Society of Virginia Peer Review Organization.

Our committees are more active than ever before, wrestling with such complex issues as the cost of health and professional liability insurance, various legislation—federal and state, medicine/business coalitions, and the expansion of the headquarters. These are only a few examples of the Society's involvement in many areas and you will learn in more detail of specifics in the committee reports.

A highlight for me personally was the Society's participation in the Summit Conference. Out of the blue a request from the White House and State Department was directed to the Society, requesting its members to provide medical service to some 6,000-8,000 people that would be in Williamsburg for four days during the Conference. Dr. Williams and Council accepted the tremendous challenge, and for almost six weeks the headquarters staff worked with State Department personnel to accomplish our mission. Three emergency medstations were set in place, and 24 of our members staffed these facilities on a 24-hour basis. The shifts varied from 6-8 hours with two physicians manning each medical unit along with nursing and clerical assistance. Although there were no major medical problems, full resuscitation equipment was on hand. The majority of medical treatment consisted of abrasions, cuts, headaches, sprains and sunburn.

The physicians volunteered their time without hesitation and some spoke as many as three languages. The mood was one of accomplishment on the last day. The Society can be proud of its participation and all were recognized by President Reagan for a job well done.

Leadership

The past twelve months have required many key decisions by the Society leadership. Our president, Dr. Harold Williams, was on hand for guidance and direction, not always an easy task. It never ceases to amaze me how our presidents, with such busy practices, manage to devote the amount of time they do to the Society. Dr. Williams continued that tradition and served with distinction. Behind his quiet and conservative manner stands a strong leader—a man of decision. Once again our Society had the right president as the right time.

Council will have met on four occasions by the time of the Annual Meeting. They, too, are dedicated people, dealing with lengthy agendas to serve their profession and provide a great service to Virginia physicians.

An important aspect of leadership is the number of meetings your president and other society officials attend. Among these are two sessions of the American Medical Association House of Delegates, AMA National Leadership Conference, Conference of State Society Presidents, annual congressional luncheon, annual meetings of our neighboring state medical societies and component society meetings.

These are only a few examples of the meetings and conferences of an ever-growing schedule each year.

Growth

Membership growth in the Society over the past five years has been tremendous. We have averaged more than 400 new members per year during this period. The total members as of this report are 6,688. Our component societies begin meeting in September, and we expect a large number of additional new members within the next three months.

Increasing membership in the AMA remains one of our most important objectives. It is good to report that AMA membership continues to gain ground. In December 1982 we reached one of our long-time goals in achieving over 5,000 AMA members. This figure entitled the Society to an additional delegate and now brings our official representation to six.

Finances

The Society's financial picture is sound. The last dues increase was implemented five years ago, and we are very proud of the fact that Virginia dues still remain one of the lowest of all state association.

The membership can always be assured the funds are managed wisely. We are confident that your Finance Committee will once again present to the House for its consideration a balanced budget.

Headquarters

Every organization has a base headquarters and for the Society, 4205 Dover Road in Richmond is it. Although still small by some standards, our personnel of 12 in number gets the job done.

To the right is a directory of MSV departments and the staff responsible for each area. It will be published from time to time in VIRGINIA MEDICAL.

I would like to thank all members of the staff for their loyal support and express my appreciation for a job well done.

Annual Meeting

The Annual Meeting will be held this year at The Homestead for the second time in the history of the Society. The meeting facilities are exceptional, and the American plan provides three gourmet meals each day.

We expect excellent attendance, and the Program Committee has devoted many hours in planning well-balanced scientific sessions as well as topics that concern us all in the field of socioeconomics.

We are very pleased to have participation from our specialty societies, which will conduct business meetings during scheduled breakfasts and luncheons, in addition to scientific programs.

Virginia Medical

For the third time in recent years VIRGINIA MEDICAL has won an award in the field of medical journalism competition. Many factors are involved in the judging, but you can be assured that outstanding appearance and content are the top criteria. It continues to be rated one of the finest state society publications in the nation and the credit, along with our sincere thanks, belongs to Mrs. Gray, Dr. Edwin L. Kendig, and the Editorial Board.

The Future

In last year's report I mentioned household words in the medical profession: FTC, HMOs, PRO and competition, for example. This

Whom to call for what at MSV headquarters

Address change	**Nancy Renalds**
Annual Meeting	
Scientific program	**Kinloch Nelson, MD**
Hotel, meeting rooms	**Mary Candler**
Blue Cross Blue Shield	**Dick Immel**
Board of Medicine	**Will Osburn**
Continuing Education	**Kinloch Nelson, MD**
CME credits	**Evelyn Stockmar**
Delegates (state, AMA)	**Mary Candler**
Dues payment	**Nancy Renalds**
Impaired physician program	**Will Osburn, Margaret Brown**
Insurance	**Dick Immel**
Legislation	**Will Osburn**
MSV Auxiliary	**Donna Strawderman**
Meetings (state, national)	**Evelyn Stockmar**
Membership (state, AMA)	**Dick Immel**
Officers (component, specialty)	**Mary Candler**
VaMPAC	**Doug Douglas, Jo Parkin**
VIRGINIA MEDICAL	**Ann Gray**
Advertising	**Frances Brown**
Travel programs	**Margaret Brown**
Risk management seminars	**Kinloch Nelson, MD**
Virginia Allergy Society	**Donna Strawderman**
Virginia Society Ophthalmology	**Donna Strawderman**
Virginia Society Otolaryngology	**Donna Strawderman**

Medical Society of Virginia 1982-1983 Officers and Councilors

President	**Harold L. Williams, MD,** *Newport News*
President Elect	**C. Barrie Cook, MD,** *Fairfax*
Past President	**H. C. Alexander III, MD,** *Roanoke*
First Vice President	**Harry C. Kuykendall, MD,** *Alexandria*
Second Vice President	**Frederick K. McCune, MD,** *Virginia Beach*
Third Vice President	**H. Dale Sponaugle, MD,** *Portsmouth*
Speaker of the House	**Richard L. Fields, MD,** *Fairfax*
Vice Speaker	**William H. Barney, MD,** *Lynchburg*
Councilors	1st District: **William Stewart Burton, MD,** *Nassawadox* 2nd: **Russell D. Evett, MD,** *Norfolk* 3rd: **James A. Shield, Jr., MD,** *Richmond* 4th: **H. Alan Bigley, MD,** *Petersburg* 5th: **Glenn B. Updike, Jr., MD,** *Danville* 6th: **William W. S. Butler, III, MD,** *Roanoke* 7th: **Robert C. Green, Jr., MD,** *Winchester* 8th: **Nicholas G. Colletti, MD,** *Woodbridge* 9th: **J. Thomas Hulvey, MD,** *Abingdon* 10th: **Leon I. Block, MD,** *Falls Church*
Vice Councilors	1st District: **William H. Sipe, MD,** *Newport News* 2nd: **John L. Dobson, MD,** *Virginia Beach* 3rd: **C. M. Kinloch Nelson, MD,** *Richmond* 4th: **John W. Hollowell, MD,** *Portsmouth* 5th: **Gerald C. Burnett, MD,** *South Boston* 6th: **Robert L. A. Keeley, MD,** *Roanoke* 7th: **John A. Owen, Jr., MD,** *Charlottesville* 8th: **Ira J. Green, MD,** *Alexandria* 9th: **James L. Patterson, Jr., MD,** *Pulaski* 10th: **Donald S. Thorn, MD,** *Annandale*
Councilors Ex Officio	**James B. Kenley, MD,** *Richmond,* State Commissioner of Health **Edwin L. Kendig, Jr., MD,** *Richmond,* Editor, VIRGINIA MEDICAL
AMA Delegates	**Michael A. Puzak, MD,** *Arlington* **Carl E. Stark, MD,** *Wytheville* **W. Leonard Weyl, MD,** *Arlington* **F. Ashton Carmines, MD,** *Newport News* **John A. Martin, MD,** *Roanoke* **Raymond S. Brown, MD,** *Gloucester*
Alternates	**George M. Nipe, MD,** *Harrisonburg* **Percy Wootton, MD,** *Richmond* **Charles M. Caravati, Jr., MD,** *Richmond* **William J. Hagood, Jr., MD,** *Clover* **Arthur A. Kirk, MD,** *Portsmouth* **H. C. Alexander III, MD,** *Roanoke*
Executive Vice President	James L. Moore, Jr.
Emeritus	Robert I. Howard

THE MEDICAL SOCIETY OF VIRGINIA • 4205 Dover Road • Richmond, Virginia 23221 • (804) 353-2721

year we add new terms: PPO, DRG and IPA. Although we understand the basic fundamentals of all, will they indeed be the ultimate solution? You and I have heard all our lives the expression, "You must change with the times." Change is good and in most instances, for the better, but not always.

In the light of approaching problems, we must remain watchful and prepare ourselves for any eventuality. Since the beginning of time, the strong survive and the weak are overcome.

James L. Moore, Jr.,
Executive Vice President

Annual Reports of Committees

Aging

Since our last report a year ago, we have had two meetings of the Committee on Aging, one on December 9, 1982, the other, June 2, 1983.

At the December meeting, Peter H. Rudy, director of Medicaid Fraud Investigation Unit of the Attorney General's office, reported on efforts of his office to investigate Medicaid fraud. His unit deals with provider fraud only, the definition of fraud being overpayments and abuse, ping-ponging from doctor to doctor, over-subscribing and billing for services not provided. He estimated that Virginia had approximately a 5% fraud rate; most appeared to be in the nursing home area, followed by physician and pharmacy violations, as in unit-dosage substitutions. Dr. Henry K. Silverman, chairman of the Geriatric Committee of the Neuropsychiatric Society of Virginia reported on his efforts to provide consultation of psychiatrists for nursing homes or any other projects which need such consultation services.

At the June meeting, Mrs. Lelia B. Harper, Deputy Secretary of Human Resources, reported on the Long-Term Care Council. She reviewed the history of the council, which primarily was formed to coordinate impaired elderly services and to identify those needing help in local areas. The goals of the council are 1) to treat the patient in-home as long as possible; 2) cost control; 3) provide appropriate long-term care locally; 4) to encourage private sect involvement; 5) provide information on services available to the patient.

Also discussed was the possible repeal of the 60-day mandatory physician visitation in nursing homes. Apparently, efforts are being made to repeal this requirement at the federal level.

Donald T. Faulkner, MD,
Chairman

AMA Delegates

The 138th Annual Meeting of the American Medical Association was held in June, and six delegates and six alternate delegates from The Medical Society of Virginia attended. In addition, the President, President-Elect and Immediate Past-President met with your delegates to argue and debate on 147 resolutions and 74 Board of Trustees and Council reports.

A new hospital medical staff section with 696 representatives of hospital medical staff met for the first time and elected officers along with an AMA delegate.

The JCAH Manual for Hospitals was extensively debated and the following Principles were affirmed: continuance of the title "medical staff"; delete reference to limited licensed practitioners; consider qualified licensed practitioners in accordance with state law; executive committee of medical staff be composed of members, the majority to be fully licensed physician members; assure that all medical care continues under the supervision of licensed physicians; and continuance of high quality care with effective quality assurance under the direction of fully licensed physicians.

Council on Medical Service Report D brought a thorough, extensive and comprehensive discussion of the many problems of payment for physician services—indemnity versus UCR reimbursement. The Council suggested that an indemnity system of payment would be beneficial to patients, and this topic will be debated in our meetings in order that your delegates will be well prepared for the AMA interim meeting in December.

Another issue of importance was the approval of a voting resident member to serve on the Board of Trustees for a two-year term and elected by the House of Delegates. In addition, a non-voting student member will be appointed to serve on the Board of Trustees.

The House adopted a policy on therapeutic drug substitution—"that the AMA vigorously oppose any concept of therapeutic substi-

At the AMA's annual meeting in Chicago in June; the President of the United States greets Dr. Michael A. Puzak, chairman of The Medical Society of Virginia's AMA delegation (left); at rear, Dr. Harold L. Williams and Dr. F. Ashton Carmines.

tution of drugs by pharmacists".

Extensive testimony and debates on the DRG program took place in the reference committees and in the House. Many problems are anticipated in this relatively untried system to be used nationwide, and plans for educational programs to clarify the details of the massive payment system are being developed by AMA. Congress has directed a study in 1984 of DRGs for physician reimbursement.

President Reagan addressed the House of Delegates on Thursday, June 23. He stated that the quality of American medicine is unsurpassed; however, the President emphasized that the cost of health care has escalated faster than anticipated and said that a one-year freeze on Medicare physician reimbursement and copayments for Medicaid may be necessary.

Michael A. Puzak, MD,
Chairman

Child Health

The purpose of the Child Health Committee is to consider all aspects of child health in Virginia. Particular emphasis is placed on legislation involving child health, welfare and safety.

A meeting of the Committee is planned prior to the Annual Meeting at the Homestead in November.

Jefferson D. Beale, Jr., MD,
Chairman

Education

The Education Committee met in Richmond on July 22, 1983, and welcomed the new members, Dr. David Propert, representing Eastern Virginia Medical School, and Dr. Anthony Munoz.

The Accreditation Council for Continuing Medical Education (ACCME) has developed "Essentials and Guidelines for Accreditation of Sponsors of Continuing Medical Education" and "Procedural and Administrative Information" which have been approved by its seven parent organizations. These go into effect January 1, 1984.

Essential #7 is taking a much firmer position on co-sponsorship by stating: *The sponsor shall accept responsibility that the Essentials are met by educational activities which it jointly sponsors with non-accredited entities. The sponsor shall 1) provide evidence that it participates integrally in the planning and implementation of each jointly sponsored CME activity; 2) conduct an evaluation of each jointly sponsored activity.*

The ACCME is working out a protocol for the recognition of state medical societies to accredit intrastate CME sponsors. Meanwhile, The Medical Society of Virginia is the accrediting body for organizations and institutions in Virginia. Council has placed this responsibility on the Education Committee.

The accredited organizations in Virginia are:

American Cancer Society, Virginia Division, Richmond
Community Hospital of Roanoke Valley, Roanoke
Eastern Virginia Medical School, Norfolk
Fairfax Hospital Association, Falls Church
Lewis-Gale Consortium, Salem
Medical College of Virginia/VCU, Richmond
Medical Society of Virginia, Program Committee, Richmond
Memorial Hospital, Danville
Northern Virginia Consortium for CME, Alexandria
Potomac Hospital, Woodbridge
Riverside Hospital, Newport News
Roanoke Memorial Hospital, Roanoke
St. Luke's Hospital Consortium, Richmond
University of Virginia, Charlottesville

Virginia Beach General Hospital, Virginia Beach
Virginia State Department of Health, Richmond
Winchester Memorial Hospital, Winchester

CME Requirement Compliance

1. In the first run of the entire membership, 1979-1981, three resigned; five or more retired; and ten were dropped.

2. In the second run for calendar years 1980-1982, of those whose last names begin with the letters A through G, there were 1,981 members. As of July 15, 30 were delinquent; several more refused to report or had not complied.

All delinquents will be reviewed by the Membership Committee prior to January 1, 1984.

Central Registry of Meetings

This continues to be active but could be greatly expanded. Those planning CME programs are urged to notify the CME office at The Medical Society of Virginia, 4205 Dover Road, Richmond, Virginia 23221, telephone number 804/353-2721, so that their topics and dates can be listed. Many conflicts could be avoided.

Annual Meeting Program

The committee is helping the Program Committee in the development of this program.

Risk Management Programs

In association with the St. Paul Fire and Marine Insurance Companies and our component societies, we have held 17 such programs covering all areas of the state except Southwest Virginia. More than 1,900 physicians have attended these. An eighteenth seminar is being considered for November 2, 1983, at The Homestead just before the Annual Meeting; more than 250 members have indicated an interest in attending such a program.

With the cooperation and assistance of Mr. Robert N. Baldwin, Executive Secretary, Mrs. Kathy Rice of the Supreme Court of Virginia, and the St. Paul Companies, we are studying the medical malpractice review panel system to determine its effects on malpractice claims in Virginia. As of December 31, 1982, 155 panels had been withdrawn, 44 dismissed, 299 concluded, and 123 were pending.

T. Eugene Temple, Jr., MD,
Chairman
Kinloch Nelson, MD,
CME Coordinator

Ethics

The Ethics Committee is gratified to report that it receive no inquires or complaints during 1983 and so was not obliged to meet.

Anthony J. Munoz, MD,
Chairman

Highway Safety

Recommendations

1. That The Medical Society of Virginia go on record as supporting legislation which would require that every person riding in any private passenger motor vehicle registered in Virginia and manufactured subsequent to January 1, 1968, be properly secured in that vehicle's child safety seat, seat belt and/or shoulder harness device.

Automobile accidents kill and injure more people in this Commonwealth than many of the diseases for which we express so much concern. The use of safety seats for children and seat belts/shoulder harnesses for adults could save a majority of the lives currently lost.

2. That The Medical Society of Virginia go on record as supporting the removal of the following phrase from all existing legislation dealing with automotive safety, such as the mandatory installation of seat belts in motor vehicles and the use of child restraint devices: "Violations of the provisions of this article shall not constitute negligence per se; nor shall violation of this article constitute a defense to any claim for personal injury . . . or recovery of medical expenses for injuries sustained in any motor vehicle accident."

Removal of this phrase from current legislation would provide incentive for people to use their car seats appropriately for their children and use seat belts/shoulder harnesses for themselves.

3. That The Medical Society of Virginia go on record as requesting the following changes be made in the Code of Virginia, Title 46.1, Chapter IV, Article 9.1, Child Restraint Devices:

Section 46.1-314.2 be amended to read, "Any driver of any private motor vehicle registered in Virginia and manufactured subsequent to January 1, 1968, shall insure that any child or ward under the age of 4, being transported therein . . . secured in a child restraint device of a type which meets current federal safety standards. . ."

Section 46.1-314.3 be amended to read, "The driver of any vehicle transporting any child so exempted shall carry on his person. . ."

Section 46.1-314.5, Sub-section B, be deleted.

There are still a significant number of children who are not being adequately protected in automotive safety devices. Furthermore, because of the difficulty in proving that the driver of the vehicle is the parent or legal guardian of the child in question, enforcement of this statute through the courts has been extraordinarily difficult, enforcement by the police has been largely nonexistent, and there is little incentive to obey the law. In states where the law is more concisely written and better enforced, there has been a noticeable drop in the deaths and injuries of children in

At a meeting of the Advisory to the Virginia Hospital Association: left to right, front row, William H. Green, Jr., of the Fauquier Hospital, Warrenton, president of the Association, Gerald J. Fisher, MD, Alexandria, chairman of the Committee, and John F. Harlan of the University of Virginia Medical Center; back row, Earl Willis of the Virginia Beach General Hospital, Edwin J. Harvie, Jr., MD, Danville, William E. Painter, MD, Lynchburg, and T. Winston Gouldin, MD, Norfolk.

motor vehicle accidents. In Virginia, this has not ocurred nor will it occur until the law is strengthened so that enforcement is feasible.

4. That The Medical Society of Virginia go on record as supporting the recommendation of the Governor's Task Force to Combat Drunk Driving. Further, that the Society and its members actively work to foster implementation of these recommendations by legislative or other means as appropriate.

The Governor's Task Force has considered the problem of the alcohol-impaired driver at length, developing 51 specific recommendations which attack the multiple facets of this problem. We believe these recommendations deserve our support.

5. That the Department of Transportation Safety and its director, John Hanna, be commended for the excellent work it has done over the last several years to promote and insure the safety of the citizens of the Commonwealth on its roadways and that the Society go on record urging the Commonwealth to maintain adequate funding for the work of this agency.

We were disappointed to see the agency lose its independent status and make this recommendation to urge that the Division of Motor Vehicles maintain the same level of interest in transportation safety that was contained in the Department of Transportation Safety.

These five recommendations were developed by the committee at its meeting in July and approved unanimously. It is our hope that the Society will consider them carefully and give them their strong support.

Joseph R. Zanga, MD,
Chairman

Hospital Advisory

Members of the Advisory Committee to the Virginia Hospital Association met with their counterparts of the Virginia Hospital Association on May 3, 1983, after a year's lapse with no formal contact. The magnitude and number of issues of mutual concern consumed several hours of deliberation and discussion and when it came to adjournment there were enough remaining unfinished items that both groups agreed to an unprecedented second session to be held before the Annual Meeting.

A legislative update by Allen Goolsby, the Society's counsel, and Nancy J. Heck of the VHA encompassed DRGs, PPOs, licensure of allied health professionals and Blue Cross territorial boundaries. The group approved the concept of hospital voluntary certification programs as an alternative to licensure for allied health professionals.

The subject of peer review for Medicare is awaiting guidelines from the Health Care Financing Administration. The State Department of Health had not yet reissued a revision of certificate of need regulations, after encountering strong opposition to its original proposals.

It was most gratifying to the chairman to have the support and cooperation of Drs. Edwin J. Harvey, William E. Painter and T. Winston Gouldin as members of the Committee. Their enlightened and articulate contributions to the discussions served The Medical Society of Virginia in good stead. The contributions of Will Osburn, James L. Moore, and Allen Goolsby were invaluable and greatly appreciated.

Gerald J. Fisher, MD,
Chairman

Insurance

The Insurance Committee has met once this year and plans to meet again before the Annual Meeting at the Homestead.

The Committee continues to review and monitor the activities and experience of all of The Medical Society of Virginia's insurance programs; however, interest in the sponsored health insurance plan with Blue Cross Blue Shield and the professional liability coverage offered with St. Paul Fire and Marine continues to dominate the Committee's attention. Appropriate selection of an insurance underwriter for all Society-sponsored programs, along with benefit and coverage design, remains the long-range responsibility of the Committee.

Effective January 1, 1983, a comprehensive major medical health insurance program was selected from Blue Cross Blue Shield. Included in this plan are provisions for deductibles, co-pays and a stop-loss provision, coupled with catastrophic benefits for the group. Simultaneously, the Insurance Committee initiated a utilization management program designed to monitor the Society's group experience in the Blue Cross Blue Shield plan and to report to the subscribers cost-saving activities and habits that can help keep premium increases to a minimum.

Effective July 1, 1983, the boundary which separated Blue Cross Blue Shield of Virginia (Richmond) and Blue Cross Blue Shield Southwest (Roanoke) was removed. It was anticipated that the two plans would merge; however, it appears that open competition will exist between the two operations. In consideration of renewing our contract with Blue Cross Blue Shield effective in 1984, it will be necessary for the Insurance Committee to examine all options, to include placing the Society's group contract out on bid to other commercial carriers.

The Society is in its 27th year of sponsorship of the professional liability product offered by St. Paul Fire and Marine. There is now evidence of strong competition from a domestic carrier as well as an out-of-state underwriter. This competition is felt to be healthy. St. Paul announced (effective with renewals on and after July 1, 1983) an average increase of approximately 13% in base rates and an additional 2½% increase for those physicians with excess limits coverage. A further refinement of this average increase indicates that premium adjustments range from a 10% reduction in rates (Class 3 Physician, Richmond Territory) to a 43% increase (Class 2 Physician, Norfolk Territory). In light of other states' experience, Virginia physician and surgeon rates and corresponding increases is not too extreme. The Insurance Committee plans to review the St. Paul territorial rating bases for the State of Virginia in order to justify their continued appropriateness. The first accounting of the retrospective total return pricing arrangement with St. Paul will occur soon after December 31, 1983.

The Society's sponsorship relationship with the David A. Dyer & Associates agency continues to flourish. The agency has made some favorable changes and additions to its insurance offerings to Medical Society of Virginia members.

There are no formal recommen-

At a meeting of the Highway Safety Committee, from left: Dr. Joseph R. Zanga, Richmond, chairman; Dr. Thomas Marvin Stiles, Newport News; Dr. David K. Webster, Staunton; and Dr. James C. Andrews, Charlottesville.

At a meeting of the Maternal Health Committee: front row from left, Dr. Fay O. Redwine, Richmond; Dr. John Q. Hatten, Newport News; Dr. William N. Thornton, Jr., Charlottesville; and Dr. John R. Talbert, Waynesboro. Back row from left, Dr. Lucien W. Roberts, Jr., chairman, South Boston; Dr. Mason C. Andrews, Norfolk; Dr. Charles Y. Davis, Blacksburg; Dr. Edward E. Gahres, Alexandria; and Dr. Bertram L. Warren, Jr., Virginia Beach.

dations to the House of Delegates, except to encourage suggestions from the entire membership concerning Society-sponsored insurance programs.

Alvin E. Conner, MD,
Chairman

Joint Practice

Recommendation

The Boards of The Medical Society of Virginia and the Virginia Nurses Association should work together to contact respective districts to ask groups to meet and express local concerns and help resolve conflicts.

General

This committee works with a similar committee from the Virginia Nurses Association to solve problems of mutual interest to nurses and physicians and help improve the quality of patient care by close cooperation of nurses and physicians in old and new forms of presenting medical care.

We had two meetings during the year and dealt with the concepts of primary nursing, medical technicians, nurse recruitment, nurse retention, nursing shortage and distribution, home health care and third-party orders.

Charles H. Townes, MD,
Chairman

Long-Range Planning

The Long-Range Planning Committee has met on one occasion this year, and in all likelihood will hold an additional meeting prior to the annual session. As directed by the House of Delegates, the Committee has under study an addition to the headquarters building, and plans for the addition have been approved by the Committee. It is hoped that a more comprehensive report can be presented in November.

William J. Hagood, Jr., MD,
Chairman

Maternal Health

Recommendation

The Maternal Health Committee recommends that The Medical Society of Virginia endorse the following nurse-midwife resolution that was approved at the spring meeting of the Virginia Obstetrical and Gynecological Society and endorsed by the State Perinatal Services Advisory Council at its last meeting, and the Committee requests that the Virginia Board of Medicine be advised of the Society's endorsement:

Resolved, that the Virginia State Board of Medicine should license nurse midwives to practice only under the direct supervision of a qualified obstetrician-gynecologist who has full active staff privileges in a JCAH approved hospital in the same community that the midwife practices. Should such an association terminate, the license of the midwife should be temporarily suspended until another association with a qualified obstetrician-gynecologist is established.

The Virginia Obstetrical and Gynecological Society was deeply concerned over the lack of proper supervision of nurse-midwives in some areas of the State of Virginia. In some instances nurse midwives were also taking care of newborn infants without supervision. The Society also recommended that the Virginia State Board of Medicine obtain the recommendations of the Virginia Pediatric Society concerning this practice.

General

At its July meeting, the Committee heard the Statewide Perinatal Services Plan reviewed by Dr. Hunsaker. This plan identifies the deficiencies and gaps in health services for Virginia's mothers and newborns and sets forth recommendations to improve Virginia's perinatal health status through the development of a formal, coordinated regionalized system of perinatal care. This plan, presented to the Board of Health at its May 18th meeting, is to serve as an advisory document for the Department of Health. The Division of Family Health Services, Bureau of Maternal and Child Health, is responsible for its implementation. Funds to implement it have been included in the Department of Health budget for 1984-86.

An update of maternal and child health activities was presented by Dr. Linyear. The 1982 session of the General Assembly allocated funds for hospitalization of infants in intensive care units and high-risk maternity care. Funds to augment maternal and infant programs have also been targeted to eight health districts where the low birth-weight rate is higher than the state rate.

A maternal PKU program has been initiated, and all known adolescents have been contacted to counsel them on getting back on the diet prior to pregnancy to prevent mental retardation in their offsprings. The issue of PKU should be brought to the attention of physicians. The newborn screening program will be expanded effective July 1, 1984, to include galactosemia, homocystinuria and maple syrup urine disease.

The 1983 General Assembly has requested that the Governor designate Healthy Mothers/Healthy Babies month. The Department of Health will conduct informational programs stressing the importance of good prenatal care.

The 1981 maternal deaths were presented to the committee. A discussion was held as to the importance of investigating maternal deaths, the information to be used for educational purposes. In the future a member of the committee will review, summarize and critique each case. The summaries will be sent to a subcommittee for review. These summaries will then be reported at the annual meeting by the Maternal Health Committee and a review will be presented at the fall meeting of the Virginia Obstetrical and Gynecological Society.

Lucien W. Roberts, MD,
Chairman

At a meeting of the Medicine/Business Coalition: left to right in the front row, Dr. G. Edward Calvert, Lynchburg, Dr. Edward A. Zakaib, Richmond, chairman, and Dr. R. L. Sallade, Newport News; second row, Dr. Robert J. O'Donnell, Falls Church, Dr. Russell D. Evett, Norfolk, and Dr. Gervas S. Taylor, Jr., Norfolk; back row, Dr. J. P. Anderson, Waynesboro, Dr. J. Latane Ware, Richmond, and Dr. Earl K. White II, Hampton.

Medicine/Business Coalition

Recommendation

That The Medical Society of Virginia endorse the establishment of a clearing house for information regarding alternative systems for health care delivery and that consideration and favorable approval be given to allocating funds for implementation of this program.

General

The principle function of this Committee is to foster the development of coalition activities between physicians and leaders of the business community. It is through this local alliance that potential solutions might be found to the increasing cost of health care. As a result of the Committee's statewide activities, coalition efforts have started in the communities of Norfolk, Hampton, Roanoke, Lynchburg and Fairfax County.

A national trend portends the development of alternative healthcare delivery systems in this country. This activity is seen in many states in response to the continued escalation of health care costs. Development of alternative delivery systems has started in Norfolk and the Fairfax County Medical Foundation has been formed in Northern Virginia to develop further ideas. It is also anticipated that Richmond area physicians will start an IPA/HMO in their community. During the year the Committee heard comprehensive presentations about various types of alternative delivery systems. All included changes in incentives to influence physician practice habits.

The Committee devoted special attention to the legal and antitrust implications of physician-organized and -operated alternative delivery systems (PPO, IPA, HMO). The Society's legal counsel and an antitrust attorney helped provide information on options available to physicians and gave advice on avoidance of antitrust exposure.

Because of this increasing interest in alternative delivery systems, the Committee sees the need for a resource center and has therefore made the recommendation.

Edward A. Zakaib, MD,
Chairman

Membership

Recommendation

That Harold L. Williams, MD, President of The Medical Society of Virginia, be nominated for honorary active membership.

Another recommendation, having to do with a resident physician classification of membership, originated in the Membership Committee but will be presented by the Bylaws Committee.

General

The Committee met twice during the year and an additional meeting is planned prior to the Society's annual meeting.

The Committee spent extensive time formulating a policy for the creation of a resident physician classification of membership in The Medical Society of Virginia. A letter and questionnaire were sent to 10% of the resident physicians in Virginia enlisting their interest.

The Committee, in an effort to enhance membership recruitment activities, set up a plan that provides free life insurance to new members of the Society. At minimal cost to the Society, each new dues-paying member is offered free term-life insurance for the first year of membership. This insurance is offered through the David A. Dyer Insurance Agency and is underwritten by the Insurance Company of America.

The Committee also reviewed the new dues-exempt policy put into effect by AMA on January 1, 1983. Although the current policy of The Medical Society of Virginia differs from the new AMA policy, the Committee prefers to allow the Society's policy to stay in effect for the time being. The Committee also reviewed the AMA policy establishing the hospital medical staff section at the national level.

J. Thomas Hulvey, MD,
Chairman

Negotiations

Recommendation

That the Negotiations Committee continue on a standby basis and that training programs be established for the purpose of maintaining a group of trained negotiators to serve the Society when needed.

General

During the past year the Negotiations Committee has been inactive, awaiting direction from the Council. The policy guidelines and procedures for the use of the Negotiations Committee by The Medical Society of Virginia through its Council were approved in 1980. The Committee stands ready to represent The Medical Society of Virginia when directed by the Council. It would appear that the constant encroachment on interests of the medical profession presents areas in which the negotiation process would be of use. Examples of such areas are many and include individual practice associations, health maintenance organizations, preferred provider organizations, Medicare, Medicaid, Blue Cross Blue Shield and other third-party organizations in which The Medical Society of Virginia does not at present formally have a part in negotiating problems of vital concern to the medical profession.

L. W. Hulley, Jr., MD,
Chairman

Pharmacy

No pressing business has arisen in the past twelve months pertaining to the Pharmacy Committee, which therefore has not met.

Potential problems continue to appear in both the state and the nation relative to pharmacists being allowed to diagnose and prescribe without medical professional oversight. An article relating to this was published in the May 20 issue of *American Medical News* entitled "Pharmacists eye study on prescribing patterns." The study referred to is a five-state study by Alan McKay, a registered pharmacist and a professor at Mercer University Southern School of Pharmacy in Atlanta.

His one-month study, the article says, showed that physician's assistants (PAs) prescribe, continue, or suggest 1.3 drugs per patient while the average for physicians is one drug. PAs may prescribe more because they "may perceive themselves as fulfilling the role of a physician only when they are prescribing medications," says the article, quoting from McKay's study. As for the drugs they prescribed, McKay's study says that central nervous system agents made up 25%, anti-infection agents about 19%, and 10% are of the "antihistamine, expectorant, cough and cold" remedies category. Of the last two groups of drugs, McKay points to their relative safety, wide therapeutic indices and low incidence of side effects and then comments, according to the report, that since experience shows that PAs handle these drugs well, even though they do not have extra training in pharmacology, why not let trained pharmacists prescribe since they are drug experts. A pharmacist with a doctorate (PharmD) already is trained in physical assessment, therapy and other essentials, so only the law needs to be changed in many states, McKay added, the article reports.

There is the additional issue of patient drug information forms to be dispensed to patients with each drug and whether there is merit in having physicians pass on information to their patients in this manner on an elective basis through the current available AMA forms.

Pharmacy issues will be reported as they develop.

Lloyd T. Griffith, MD,
Chairman

Physicians' Health and Effectiveness

Recommendations

1. That an appropriation in the amount of $12,000 be made for the operation of the Committee during 1982-1983 and that Council be authorized to appropriate additional funds should it be necessary.

The House of Delegates appropriated $10,000 for the current year of which approximately $9,300 has been spent. The major portion of this year's expenditure was for meetings, workshops, and preparation and mailing of 28,000 brochures. Since the actual program is just now getting under way the financial needs for next year are difficult to project. The committee does anticipate an annual workshop for investigators and intervenors and it is possible that additional educational materials may be needed.

2. That the Society pay intervenors a fixed mileage rate or distance travelled and out-of-pocket expenses for meals and lodging necessary to their duties.

The Medical Society of Virginia has not in the past compensated officers or committee members for their services with the exception of the President. Committee members can claim mileage but rarely do so. Intervenors, however, are limited in number and in many cases will have to travel considerable distance, perhaps several times for each intervention. The Committee recognizes that the society cannot possibly compensate a physician for time away from practice, but it feels that actual expenses should be reimbursed. It is anticipated that this item will be covered by the annual appropriation requested above.

General

The committee has held three meetings so far this year, in February, May and August. The May meeting was in conjunction with a day-long workshop for intervenors and investigators conducted by Dr. Douglas Talbott. Approximately 60 members attended.

The Impaired Physicians Program actually became operational on July 1st. A number of reports have been received and investigations instituted. The Committee has decided, upon advice of legal counsel, that it will not now release information relative to the number of cases reported or their disposition. The committee recognizes that the membership of the Society is entitled to have, from time to time, statistical information in order to examine the success of the program. This will be achieved by regular reports to Council and through VIRGINIA MEDICAL.

There are three items which the committee has under continuing study which will require House action sometime in the future.

Education. The Committee is committed to an extensive educational program. The beginning of this has mainly been the brochure, scattered appearances of committee members before Society and Auxiliary meetings and media coverage. Action is being initiated in several other areas.

The Committee has met with representatives of the three Virginia medical schools and all are beginning or enhancing existing programs to expose medical students to the problems of chemical abuse. The Committee has offered to help them in anyway that they desire. Specifically, it is suggested that the Society provide teaching aids, films, publications, and make available members of the Society who are qualified to lecture on the subject.

Committee members and intervenors will be available, as time allows, to speak at component society and Auxiliary meetings to explain the program.

A study is under way to determine the feasibility of a narrated slide presentation describing the Virginia program which can be used by societies and Auxiliary.

The Committee has received an offer of funds donated by another organization, which can be used over and above the basic budget. These funds, if applied to education, could enlarge substantially our informational impact.

Staffing. Other states have found that as the program grows, permanent staffing is a necessity. The Committee has voted to use present Medical Society Virginia staff for the next 6-12 months as we evaluate such needs. It appears likely that a non-professional staff member, capable of programming intervention and available for educational purposes, will be needed by 1984, and that a medical professional could be necessary by 1985.

Loan Fund. Programs in other states have found it advisable to establish a loan fund to provide hospitalization for physicians who cannot pay for such institutionalization. There are a number of ways to establish such a fund, one being a moderate assessment of members over a 2-3 year period. Council was authorized last year to consider this and a sub-committee of the Physicians Health and Effectiveness Committee has been appointed to work in this area.

The last three items will have an important financial impact on the Society and members are invited to comment to the committee on these or other aspects of the program.

William H. Barney, MD,
Chairman

PSRO Liaison

With passage of the peer review organization legislation in 1982, The Medical Society of Virginia House of Delegates resolved at its last meeting that the Society should move toward becoming the peer review organization in Virginia. The Liaison to PSRO Committee recommended to Council general guidelines for the development of The Medical Society of Virginia Review Organization (MSVRO), a separate corporate entity whose board of directors will be appointed by the President of the Society. These guidelines were adopted by Council.

An ad hoc committee to organize the MSVRO consisting of the President, Immediate Past President and President Elect of the Society; one member from each congressional district; and four at-large members has been appointed by the President and, with the full cooperation of the current PSROs, is developing an organizational structure, a plan for reviewing the necessity and quality of care rendered to Medicare beneficiaries, and the necessary budget. This ad hoc committee will become the board of directors of the MSVRO upon its incorporation. It is not clear just when the federal government will implement the 1982 legislation but it is felt that The Medical Society of Virginia will be ready to keep peer review a province of practicing physicians and not let this activity go by default to any other agency.

With the submission of this report and the phasing out of PSRO, it appears that the Liaison to PSRO Committee has completed its mission. The MSVRO will continue to keep the Society informed relative to peer review activities.

Robert A. Morton, MD,
Chairman

Public Relations

The three high points of the year for the Public Relations Committee were the excellent media coverage of the initial instruction meeting of the Physicians' Health and Effectiveness Committee; the excellent statewide (plus District of Columbia) media coverage of the 1982 Annual Meeting in Williamsburg; and the nationwide coverage of our medical support effort of the Summit Conference of the Industrialized Nations at Williamsburg.

These successes are primarily due to the outstanding efforts of Ed DeBolt, Carole Cones and Doug Douglas.

It is imperative that the Society continue with the caliber of legislative personnel and public relations consultants if we are to keep our image visible.

The Committee thanks each of you who have contributed to the aforementioned efforts and those of your local societies, which have given The Medical Society of Virginia good material to build public relations upon.

Frederick K. McCune, MD,
Chairman

Rehabilitation

During the current year, the Rehabilitation Committee has contin-

ued its primary role of providing consultation and recommendations to the Virginia Department of Rehabilitative Services in regard to medical policies and procedures. The services provided by this Department have considerable impact on the medical community and on patient care, and the Committee's input to the Department is most appropriate and valuable.

The Committee met on May 22, 1983. Eleven members were in attendance. Also present were several members of the staff of the Virginia Department of Rehabilitative Services. The Committee was informed that the Disability Determination Services had noted a better understanding and more assistance from the physicians of the state in its program of mandatory periodic review of Social Security disability recipients. It was felt that the activities of the Committee during the past year contributed to this improved rapport. It heard a report on a proposed educational project to inform the physicians of the state regarding the goals and services of the Department of Rehabilitative Services. The Committee considered a number of specific diagnostic and therapeutic procedures and made recommendations to the Department.

Throughout the year, individual members of the Committee provided advice and assistance to the staff of the Department of Rehabilitative Services.

This has been an active and productive year for the Committee but there are no recommendations.

Alexander McCausland, MD,
Chairman

Rural Health

The purpose of the Rural Health Committee is to review and evaluate all rural health problems as they are brought to the attention of The Medical Society of Virginia and to advise and guide the 4-H Clubs Health Awards program.

Having no business before it, this committee reports no action for 1982-1983; thus there are no recommendations.

Harold W. Felton, MD,
Chairman

State Bar Liaison

The Liaison Committee to the State Bar held its annual meeting with a representative group of the Virginia State Bar. The role of the Committee has been to address and resolve problems of mutual interest and concern to physicians and attorneys.

The joint Committee addressed the question of fees for medical testimony in connection with the resolution adopted by the House of Delegates in 1982. This resolution calls for local and regional committees of The Medical Society of Virginia and the Virginia State Bar to work together with the judiciary to promulgate guidelines. The chairman of the State Bar group reported no response to all his letters to Richmond, Henrico and Chesterfield judges and bar associations and the Richmond Academy of Medicine. The executive secretary of the Virginia Supreme Court reported that the Executive Committee of the Judicial Conference of Virginia (Supreme Court and circuit Judges) had referred the matter to a subcommittee chairman. Evidently, the judicial circuits are grouped informally in several regions, with rotating chairmen, and the sense of feeling of these judges was that the initiative for promulgating guidelines should emanate from a joint committee of physicians and attorneys and not from the judges themselves.

The Committee was apprised of the progress and proceedings of the medical malpractice review panels as prescribed by Virginia State statute. The panels continue to be used extensively and considerable attention was focused on the appropriateness of the various health care provider members of the six-member panels. Physicians and attorneys alike have expressed concern about the meaningfulness and cost-effectiveness of panel activity. The physician members of the committee have therefore directed staff of The Medical Society of Virginia to survey the results of the panels concluded through July 1, 1983, and report back to the committee at its next regular session.

The Committee has no formal recommendation to make to the House of Delegates.

Claude P. Sherman, MD,
Chairman

VaMPAC

The Virginia Medical Political Action Committee experienced the best financial year in its 20-year history and the largest membership ever for a year in which there were no federal elections.

Three of every four candidates supported by the PAC in the last election cycle were victorious; these include both Republicans and Democrats, for we feel good candidates transcend party lines. This is a record of accomplishment of which the entire medical community can be proud. Yet we should take greater pride in what these facts and statistics actually represent—thousands of doctors and their families who have voluntarily supported the PAC with dollars as well as their valuable time. These are the people who recognize that the future of our health care delivery system is in large measure determined by today's legislative decisions.

In addition to direct financial assistance and encouragement, the PAC provided valuable political expertise as well as special assistance to warranted campaigns where the medical community's personal involvement could make the difference between winning and losing.

Here are some highlights of the VaMPAC year:

- Auxiliary members are now able to join VaMPAC directly in a membership, category due to recent court rulings. The response of the Auxiliary has been suprisingly high.
- 1983 saw the first in the series of a three-year joint fund-raising activity by AMPAC/VaMPAC in an effort to involve more of the medical community in the political process and candidate support.
- AMPAC/VaMPAC sponsored a political education seminar in September for PAC 250 members designed to increase their personal political effectiveness.
- The executive director of VaMPAC traveled throughout the state attending many component society meetings, sharing political information and ideas, encouraging grass roots support in the political process.
- VaMPAC has opened a new avenue of involvement for PAC 250 members. VaMPAC will be allocated a number of spaces by the White House for the official White House welcoming ceremonies for heads of state on the South Lawn.
- VaMPAC's presence again was felt through its director, who was instrumental in arranging for President Reagan's trip to the AMA meeting in Chicago—resulting in personal meetings and photo sessions with VaMPAC, MSV and AMA groups.
- The executive director, while taking an extended leave of absence without pay to serve as deputy director of the 1983 Summit of Industrial Nations, labored hard and long to facilitate VaMPAC activities through his involvement in the Summit operations. Many VaMPAC board members and PAC 250 members were in final attendance during the Joint Statement ceremonies.
- In January, VaMPAC held a much acclaimed reception at the Commonwealth Club the evening before the start of the 1983 legislative session to honor its leadership. Also attending were Gov. and Mrs. Charles S. Robb, Lieut. Gov. Richard J. Davis, and Attorney General and Mrs. Gerald L. Baliles.

Percy Wootton, MD,
Chairman

Vanguard

This has been a year of transition for the Vanguard Committee. Originally founded to meet the challenge of Public Law 93-641 and the HSA, the committee functioned efficiently during those years bringing to The Medical Society of Virginia news from each of the HSAs and providing the leadership of the Society an instrument which was effective in dealing with local agencies.

By unanimous vote at our July meeting it was agreed to turn our attention to the problem of prospective payments as they will relate to physicians who participate in Medicare. By January 1985, prospective payment legislation for Part B will almost certainly be enacted. It is hoped discussion and dialogue with Congress can be accomplished before hasty enactment occurs.

At the July meeting the committee was addressed by F. Lynn May, Director of External Affairs for the Health Care Financing Administration. Also in attendance was Robert Hausenfluck, Deputy Assistant Secretary of Health for Public Affairs. They outlined for the committee the basic goals of HCFA in planning this legislation for physicians. The plan, given in broad strokes only, hopes to decrease confusion about assignment of benefits, increase the simplicity of Part B reimbursement, increase physician participation, and increase the efficiency of the payment system. Lofty goals all, but not readily achievable.

In the view of many, there are no data to support the hypothesis of the Department of Health and Human Services that these things will happen. The data base for DRGs as applying to hospitals is quite narrow, and much concern exists as to how the legislation already enacted will affect hospital operations. The information on how individual practitioners will be affected by the changes in Part B legislation is open to even more question. Not only must inpatient services be covered, but the problem of payment for outpatient and extended-care facilities will also have to be addressed. Simple definitions about what constitutes an episode of physician care subject to reimbursement are difficult to conceptualize. How will the payments be made? To whom? For what? For how long? These are fundamental questions to which there are no uniform answers. An issue which is paramount in all of our minds is quality of care. That often comes at the bottom of the government's list of priorities, but Mr. May was quick to point out that physicians, not the government, are in charge of quality. Cost has been and will continue to be the driving force for prospective payment legislation whether it affects hospitals or physicians.

The Vanguard Committee, recognizing the enormous impact of these questions on our profession, will in the next few years attempt to make the views of the Society

felt in Washington. At the Annual Meeting in November, Mr. May will address the Society and provide answers for some of these important questions. The Committee urges each of you to attend the session.

J. Hayden Hollingsworth, MD,
Chairman

Virginia Medical

Somebody seems to be reading Virginia Medical. We are continuously fielding requests for extra copies of this or that issue. Despite an extra press run, the supply of the November 1982 issue on breast cancer and informed consent is now exhausted, due to the multiplicity of requests for it from all over the country. Among those who found it of merit were the judges of the Sandoz Medical Journalism competition, who gave it a special citation. This is the third award Virginia Medical has won in this contest; it is to be presented before the House of Delegates at the annual meeting in Hot Springs.

Such recognition is not to be sneezed at, but the prizes the Editors like best of all are the contributions so kindly sent to us for publication. In fact, where would we be without them? Medical articles, historical texts, editorials, commentaries, letters to the Editor—it is a privilege to share with our readers these splendid communications, and we thank heartily all those whose triumphs over the rigors of authorship enhanced Virginia Medical's pages.

Edwin L. Kendig, Jr., MD,
Editor

Sports Medicine

The Sports Medicine Committee was very active this year.

The legal status of athletic trainers was the most important subject before the Committee, which is working with the Medical Licensure Board to resolve the various issues involved and to aid in implementation of a formal certifying process. The process has taken substantial time and multiple meetings.

The Committee established liaison with the Virginia Chapter, US Olympic Committee.

There was continued liaison with the Virginia High School League. Under the leadership of Dr. Frank McCue, the Annual High School Coaches Clinic was held on July 15.

Robert P. Nirschl, MD,
Chairman

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VIRGINIA MEDICAL OBITUARY

Bruce B. Baird, MD

Dr. Bruce Douglas Baird, 39-year-old Roanoke surgeon, was drowned on August 22 while on vacation at Emerald Isle, North Carolina, in a swimming accident that also claimed the life of his father but spared his 4-year-old son.

Although the beach was crowded at the time of the tragedy, Emerald Isle police could find no witnesses to the tragedy. Dr. Baird's son, Spencer Kenneth Baird, apparently was saved from drowning by clinging to his father's body and was rescued from the surf unharmed. Dr. Baird, however, despite resuscitation efforts, was pronounced dead on arrival at Carteret General Hospital in nearby Morehead City. The body of Dr. Baird's father, W. Kenneth Baird of Oil City, Pennsylvania, was found about 100 yards offshore.

Born in Butler, Pennsylvania, Dr. Baird was graduated in 1969 from the University of Vermont College of Medicine. A veteran of the United States Navy, he was from 1975 to 1978 chief of surgery at the US Naval Hospital in Cherry Point, North Carolina. He was associate director for surgical education for all Roanoke Hospitals and assistant professor of surgery at the University of Virginia School of Medicine. He came to membership in The Medical Society of Medicine through the Roanoke Academy of Medicine.

William P. McGuire, MD

Dr. William Province McGuire, ophthalmologist and member of a family distinguished in Virginia medicine since 1801, died August 22 at his home in Winchester. He was 75 years old.

Dr. McGuire was the great-grandson of Dr. Hugh Holmes McGuire, the family patriarch who founded Virginia's first medical school, Winchester Medical College. He was the grandson of Dr. William Province McGuire, surgeon; the son of Dr. Hunter Holmes McGuire, also an ophthalmologist; and the great-nephew of another Hunter Holmes McGuire, MD, who practiced surgery in Richmond and attended Gen. "Stonewall" Jackson during his campaigns for the Confederacy.

A 1935 graduate of the University of Virginia School of Medicine, Dr. McGuire was a captain at the US Naval Hospital in Bethesda, Maryland, and in the Pacific during World War II. Elected to Winchester's city council in 1948, he served two terms.

In 1973, Dr. McGuire was elected president of the American Ophthalmology Society, a post his father also held. He also succeeded his father as president of the board of directors of Winchester Memorial Hospital, where, in 1980, a new wing was named in honor of the family. The Medical Society of the Valley of Virginia had also named him its president.

Dr. McGuire's other professional memberships included the Northern Virginia Medical Society, The Medical Society of Virginia, Southern Medical Association, American Medical Association, American Academy of Ophthalmology and Otolaryngology, and Ophthalmology Society of the United Kingdom.

Luther H. Apperson, MD

Dr. Luther H. Apperson, for 57 years a family practitioner in Richmond, died at his home on August 16. He was 93 years old and was still maintaining a limited practice.

A native of New Kent County, Dr. Apperson was graduated from Virginia Polytechnic Institute in 1909 and earned his medical degree from the Medical College of Virginia, Class of 1912. He trained at Central State Hospital, Petersburg, before establishing a practice in Southern Virginia that included Pittsylvania, Halifax and Charlotte Counties. He later practiced in Petersburg and Colonial Heights. In addition to duties as surgeon for the Atlantic Coast Line and Southern Railroads, he was associated with the Virginian Railroad from 1913 until 1926. He located in Richmond in 1926, to practice there until his death.

Dr. Apperson's name had been on the membership roster of The Medical Society of Virginia for over 70 years, and he was a longtime member of the Richmond Academy of Medicine and the American Academy of Family Physicians.

John M. Adams, MD

Dr. John M. Adams, Winchester radiologist, died at the age of 79 on February 2, 1981, at Winchester Memorial Hospital.

Dr. Adams was a native of Southern Pines, North Carolina, and was graduated from the Jefferson Medical College, Philadelphia, in 1929. He received training at Episcopal Hospital in Philadelphia, Central Islip, New York, State Hospital and the University of Virginia, Charlottesville. Between the years 1943 and 1947, he practiced in Tennessee, Ohio and Indiana, and moved to Virginia in 1947, settling in Winchester.

A member of the Radiological Society of North America and the American College of Radiology, Dr. Adams came to membership in The Medical Society of Virginia in 1949 through the Northern Virginia Medical Society.

Maurice L. Courtney, MD

Dr. Maurice Lee Courtney, Morgantown, West Virginia, psychiatrist, died May 30 at Baptist Hospital in Knoxville, Tennessee, where he was undergoing surgery as a result of an automobile accident two weeks earlier. He was 61 years old.

A Knoxville native, Dr. Courtney received his medical degree in 1951 from the University of Tennessee, Memphis. Upon completion of his internship at Knoxville General Hospital, he trained at Warren, Pennsylvania, State Hospital and Indiana University Medical Center, Indianapolis. He came to Richmond in 1970, and though he practiced there for only six years before relocating to Morgantown, he retained his Medical Society of Virginia membership. Dr. Courtney was also a member of the American Psychiatric Association and the American Medical Association.

R. M. Miskimon, MD

Dr. Robert M. Miskimon, Richmond, died August 24 at the University of Virginia Medical Center, Charlottesville, at the age of 69. He had retired in 1977 after 20 years as medical director of the Fidelity Bankers Life Insurance Company of Richmond.

A graduate of Darmouth College, Dr. Miskimon received his doctorate in medicine in 1941 from Columbia University College of Physicians and Surgeons, New York, then served in the Army Medical Corps from 1942 and 1946. Before joining the life insurance company, he was chief medical examiner of the Richmond City Employees Medical Service.

Dr. Miskimon had belonged to The Medical Society of Virginia for almost 40 years. He was a member also of the Richmond Academy of Medicine and the Industrial Medical Association.

Memoir of John W. Thomas 1909–1982

By Hassan Ali Mohagheghi, MD, Herbert H. Galston, MD and Frank R. Kelly, Jr., MD

Dr. John Warrick Thomas, Richmond internist and allergist, died in a local hospital on May 21, 1982, of coronary heart disease at the age of 73.

Born in Tennille, Georgia, Dr. Thomas attended Virginia Military Institute and was graduated from the University of Georgia and its School of Medicine. While in residency at the University of Georgia Hospital, he met and married the former Norma Busbee, a graduate nurse. Dr. Thomas completed his residency training in allergy at the Vaughan Memorial Clinic, later the Graham-Thomas Clinic, Richmond. He then served as head of the allergy department at the Cleveland, Ohio, Clinic before moving to Richmond, where he opened his own allergy clinic in 1948 and practiced until his death.

Dr. Thomas was a member of Alpha Omega Alpha, medical honor fraternity, a founder and former president of the American College of Allergists, and a member of many other national and international allergy associations.

He was a member of the Richmond Academy of Medicine, and because for many years he maintained a summer home on the Rappahannock River, he belonged also to the Northern Neck Medical Society. His other memberships included The Medical Society of Virginia, the Richmond and Virginia Societies of Internal Medicine, and the American Medical Association.

Survivors include his wife, Norma Busbee Thomas, a daughter, two sons and five grandchildren, all in Richmond.

Prepared and published at the request of the Richmond Academy of Medicine.

BOOKS

The Harvard Medical Unit at Boston City Hospital, Volume II (Part 1. The Peabody-Minot Tradition. Part 2. The Castle-Finland Era), by Maxwell Finland and William B. Castle. Boston, Commonwealth Fund Publication, Harvard Medical School, 1983, 1041 pages, $50.

In Volume I of this two-volume work, Maxwell Finland presented a history of the Thorndike Memorial Laboratory, the Second and Fourth Medical Services, and the significant events of Boston City Hospital from its founding in 1865 to 1974, including much biographical material on chiefs of services and professors, such as Peabody, Minot, Soma Weiss, Chester Keefer, and Finland and Castle themselves.[1]

This second volume is comprised of two books, one of biographies, one of autobiographies, 642 in all, of physicians who participated in the Thorndike and Harvard Medical Services from 1905 to 1974. Many of these physicians are Virginians. It would be difficult to find a more complete collection of such material.

The biographies are short and factual. The autobiographies vary from a few lines to several pages long. There is much valuable information and praise of the Thorndike and Harvard Medical Services. Many of those who wrote autobiographies went into great detail from their early school experiences, including their various professional relationships and accomplishments. It should be noted that this is the kind of information Dr. Finland asked for. He wanted to know the contributor's hobbies, and when he wrote to me, he asked if my wife and I were still riding horseback.

To quote the finis of Volume II: *The lotus lifts its lovely flower above the black muck of the swamp whence comes its beauty. So did the Boston City Hospital—lowly, drab and unappealing to the eye—nourish with the help of the Harvard Medical School, the flowering of compassionate physicians, imaginative and industrious investigators and dedi-*

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cated teachers. Together with their students and staff they pursued excellence in the care of needy sick and sought to heal their bodies and their minds. To labor in the Harvard Medical Unit was for most a special fellowship and privilege ever to be remembered.

R. Bryan Grinnan, Jr., MD

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1. Grinnan RBjr: The Harvard Medical Unit at Boston City Hospital, Volume I, by Maxwell Finland (book review). Va Med 109:819, 1982

Editor's Note: The book may be ordered from its distributor, University Press of Virginia, Box 3608, University Station, Charlottesville VA 22903. To the price of $50, add $2 sales tax and $1 for postage—total, $53. Or by donating $100 to the Charles S. Davidson Professorship Fund, Harvard Medical School, you will receive copies of both Volumes I and II. Checks should be mailed to Max Finland, MD, Boston City Hospital, Boston MA 02118.

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Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out

Precautions. *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response this drug should be used during pregnancy only if clearly needed

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor* (cefaclor, Lilly) is administered to a nursing woman

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established

Adverse Reactions. Adverse effects considered related to therapy with Ceclor are uncommon and are listed below

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70)

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients)

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40)

Hematopoietic—Transient fluctuations in leukocyte count predominantly lymphocytosis occurring in infants and young children (1 in 40)

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200)

[061782R]

* Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[4]

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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8. Principles and Practice of Infectious Diseases (edited by G.L. Mandell, R.G. Douglas, Jr., and J.E. Bennett), p. 487. New York: John Wiley & Sons, 1979

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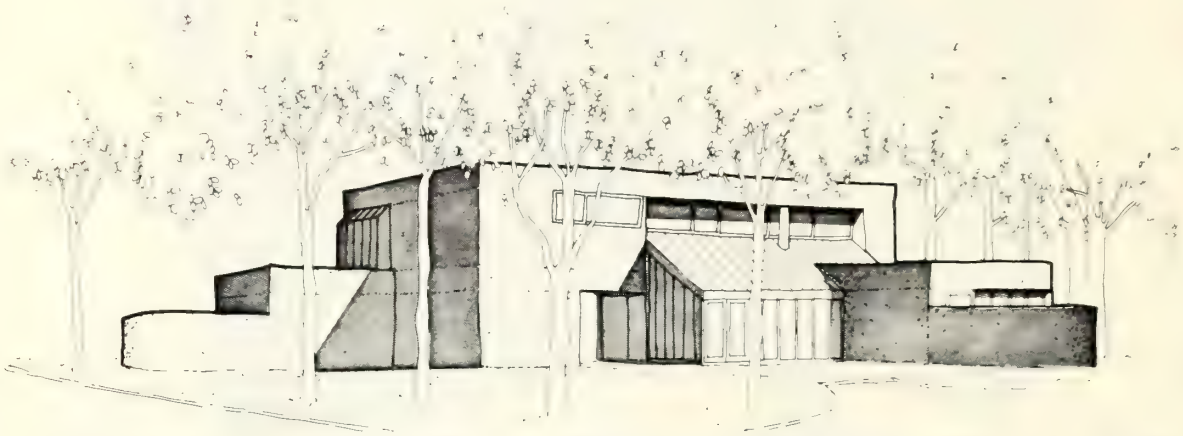

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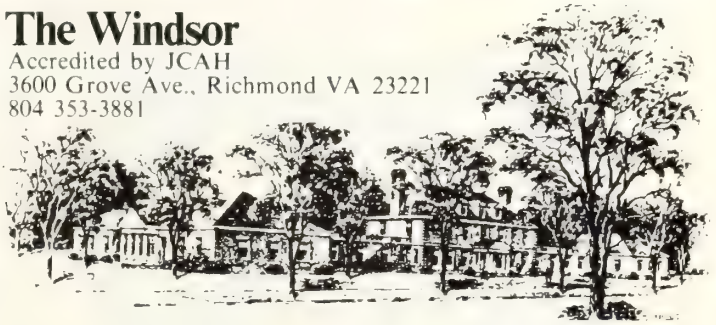

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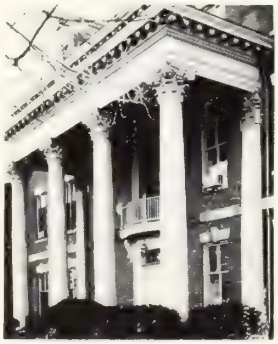

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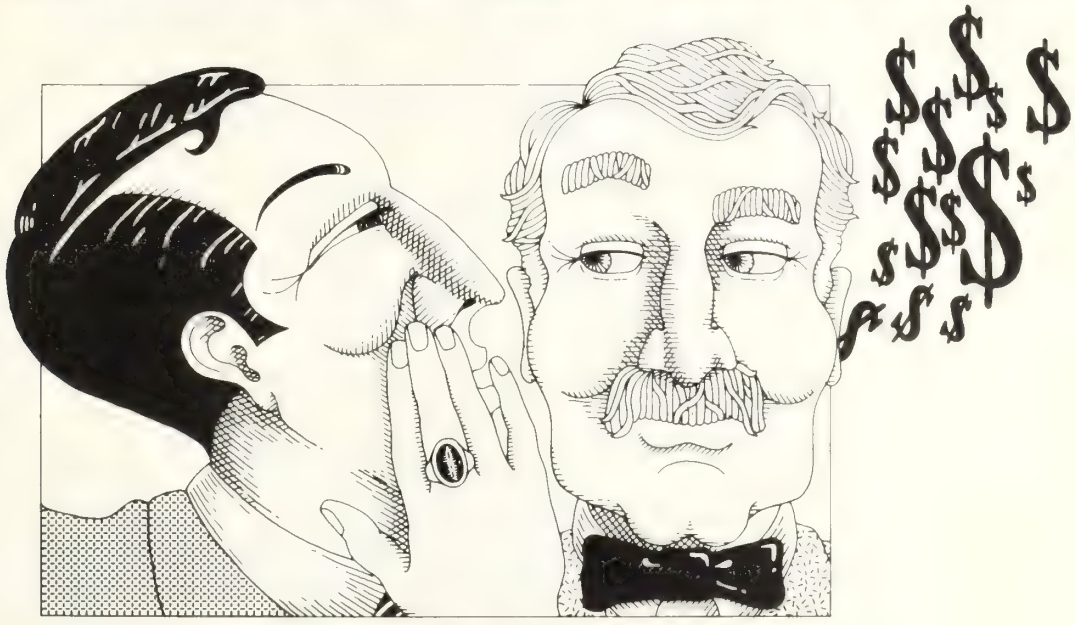

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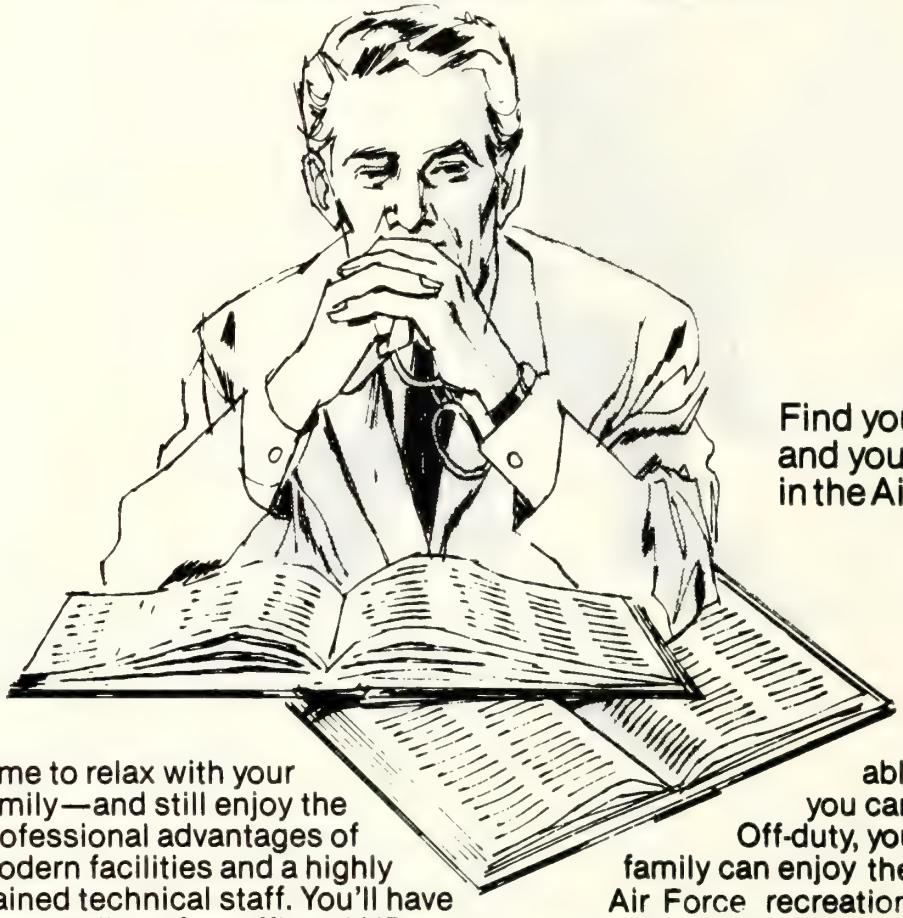

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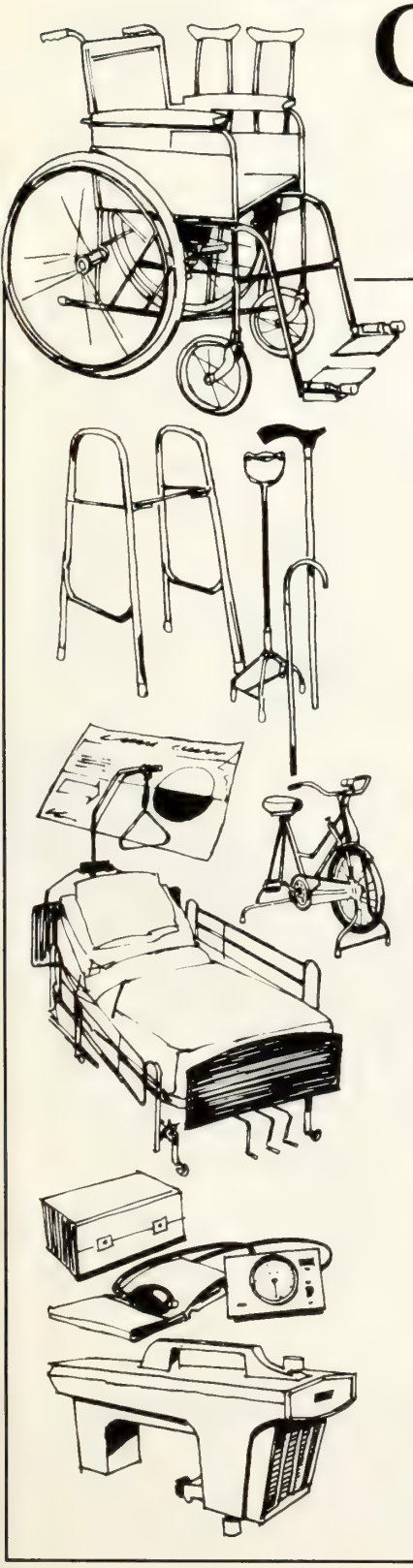

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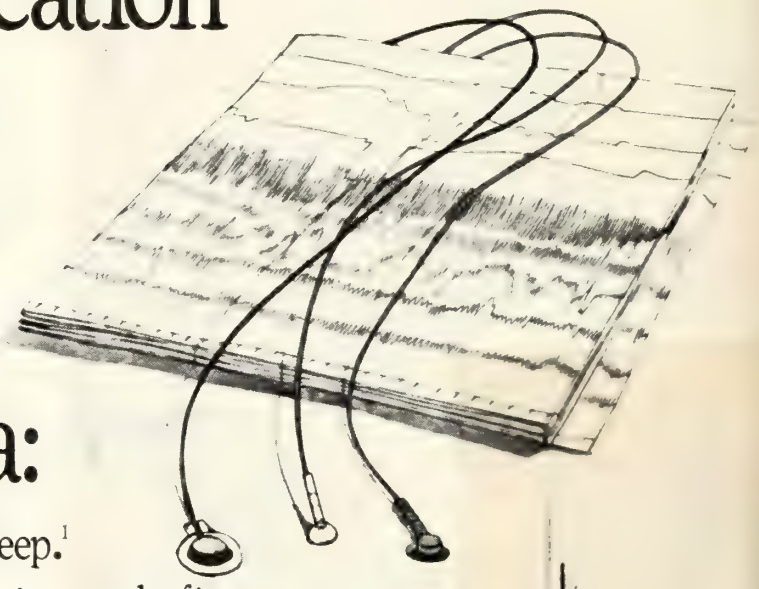
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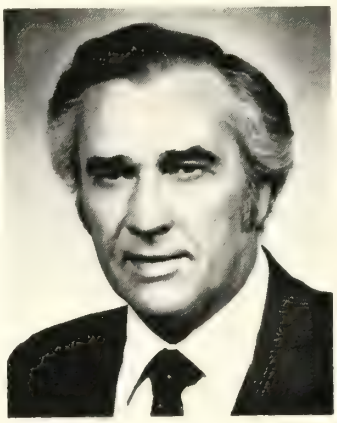

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New HMO, IPA moving into Virginia medicine

A NEW HEALTH MAINTENANCE ORGANIZATION is poised for lift-off in Northern Virginia under the auspices of a hospital corporation, and in the Tidewater area a physician-owned independent practice association is working toward a January 1 launch.

The HMO is Virginia Health Plan, Inc., a group-practice, or open-panel, type of HMO in which physicians contract to treat subscribers in their offices while continuing to conduct their own practices.

As this was written, 50 doctors had signed up, and the plan's developer was seeking 50 more. Elected chairman of the board and president was Dr. John C. Bucur, neurological surgeon who is a past president of the Arlington County Medical Society.

Ownership of the HMO rests with the National Hospital Health System Corporation, a holding company that owns National Hospital for Orthopedics and Rehabilitation in Arlington. Dr. Bucur is a director of the corporation and is chief of neurological surgery at the hospital. Management of the prepaid plan has been assigned to Jurgovan & Blair, a Rockville, Maryland, consulting and operations firm.

Relationships with several hospitals have been established, according to David L. Weiss, projects director for the plan, but the doctors under contract may admit patients to any of the hospitals they are accustomed to using. The plan is designed to deliver medical service in Arlington, Fairfax, Falls Church, Alexandria and Prince William County, Weiss said, and will offer employers several benefit and premium options.

Northern Virginia is the home of Kaiser-Georgetown, a closed-panel HMO owned by the granddaddy of all health maintenance organizations, California's Kaiser-Permanente. In 1980 there was exploration of HMO-IPA feasibility in Northern Virginia, but the project failed of consensus among physicians of the area.

THE PHYSICIAN-OWNED IPA in the Tidewater area, Health Plan of Virginia, Inc., may get off the ground early in January 1984, according to Dr. Claude A. Smith, Norfolk obstetrician and gynecologist who is the plan's president. Dr. Patrick C. Devine, Sr., Norfolk, is vice president and Dr. Leonard L. David, Jr., Portsmouth, secretary.

Like the Northern Virginia HMO, the Tidewater IPA is an open-panel system and has mapped out a broad geographical area encompassing Norfolk, Virginia Beach, Portsmouth and Chesapeake.

At the time Dr. Smith talked to VIRGINIA MEDICAL's reporter, 101 physicians had signed up; each were required to buy stock in the corporation in order to join. The goal is a membership of 500 doctors, but 125 will be sufficient for start-up, Dr. Smith said. He expects to have contracts ready to offer clients this month. A professional consultant from Bethesda, Maryland, has been in Norfolk since July drawing up management specifications.

Ann Gray wrote the stories on HMO and IPA activity. Carl L. Shires wrote the report on KeyCare.

VS BUREAU

Three Norfolk corporations have been advising the plan's founders, Dr. Smith related, and the board of directors ultimately will include both business men and consumers. The board now is comprised of 30 physicians; 19 of them, Dr. Smith observed, are former presidents of either their local medical societies or the medical staffs of their hospitals. "That's the caliber of support we have," Dr. Smith commented.

"Two years ago I would have been as opposed to this as anyone down the creek," Dr. Smith recalled. "But when I was in the hospital not long ago, I did a lot of reading up on HMOs and PPOs and IPAs, and I came to the conclusion that business is fed up, and we'd better do something about it.

"This plan is aimed at preserving, one, private medical practice as we know it; two, quality of care; three, the patient's pattern of care.

"I've been in practice for 22 years, and I don't want to see any of those things change."

A GROUP-PRACTICE HMO that began operating in Virginia late last year, PruCare of Richmond, is going great guns, in the view of doctors at

Just when you thought it was safe enough to go back in the creek. . . .

First there was Jimmy Carter's killer rabbit. Now comes Dr. Lockhart B. McGuire's brazen beaver, and the Charlottesville physician has the scars and stitches to back up his story.

Around 6 pm [on August 27], McGuire was pushing an inner tube to deeper water as he was swimming in the Cowpasture River on his farm near Williamsville in Bath County. Suddenly he felt a sharp pain in his leg.

At first McGuire thought he had been bitten by a snake, but then he reached down and found something that was "covered with fur and had a big, flat tail" stuck to his leg. McGuire managed to make it to shallow water where he found a beaver with its teeth firmly sunk in his leg.

For about a minute, McGuire struggled with the animal until he was able to pry its jaws loose from his leg. McGuire's calls for help were answered by his daughter, who arrived in time to see the beaver swimming away.

McGuire was taken to the Bath County Community Hospital, where he received several stitches. The wound, he said, showed punctures from upper and lower teeth. Since the run-in with the rodent, McGuire has begun taking rabies shots.

McGuire said he is "beginning to laugh about it" now that his leg is healing. He said colleagues have compared the attack to President Carter's encounter of several years ago with an acrimonious, amphibious rabbit.

"The game warden was surprised that it happened," McGuire said. "He thought maybe it was a mother protecting her cubs, but I didn't see any cubs."

As for the real reason for the attack, we'll have to leave that to the beaver.

—BRIAN SHAW

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the McGuire Clinic, which contracted to deliver the plan's medical care.

"It's phenomenal," one doctor exclaimed. "We have 13,000 new patients. The parking lot's jammed. We're staying open Saturdays and Sundays. They've added ten doctors. I had a Sikh patient the other day—it's an international experience!" he related so exuberantly that he skipped over his pun.

"The clinic's buzzing," confirmed another McGuire doctor. "Of course," he added, "all those patients are coming from other doctors, which isn't so good."

Of the plan's progress thus far, James W. Brittain, PruCare's vice president said, "We're ahead of projections and are very pleased with the reception in Richmond." The plan is owned by Prudential Life Insurance of America, which operates HMOs in other cities.

Subscribers to PruCare have come from such firms as Reynolds Metals, Best Products and Media General, Brittain said, and from employees of Henrico County and the State of Virginia, including the Medical College of Virginia. The goal is to enroll 80,000 members in the next ten years. Dr. R. Almond Hilton is PruCare's medical director.

What effect is Blue Cross Blue Shield of Virginia's new preferred provider organization, KeyCare, expected to have on PruCare? "Until we have some experience competing, it's difficult to say," Brittain commented, "but we certainly aren't going to take the KeyCare program lightly."

IT'S A GOOD IDEA not to take KeyCare lightly, because in its initial months the Richmond-area PPO has surged faster in both physician and hospital sign-ons than even its most optimistic advocates had anticipated.

By late September, about 700 Richmond area physicians had agreed to participate in KeyCare, and the contracts were continuing to come in at a rate of between 25 and 50 per week, Peple said. There are nearly 1,600 licensed physicians in the greater Richmond area.

Among hospitals in its bailiwick, 13 in Richmond and one in Petersburg had signed KeyCare contracts as this was written.

With the hospital participants virtually closed out and physicians and other providers still signing contracts, the next major move for KeyCare is to sign insured groups on the dotted line.

"We're optimistic on that," said Edward C. Peple, Jr., manager of KeyCare development for the Blues. "Initially, it was the old cart-before-the-horse thing. Groups were not interested until we got an adequate provider base, and providers wanted to know what groups would participate. Now we've got the provider base, and the groups will be coming in as existing contracts expire."

Meanwhile, plans for expanding the Blues' KeyCare program to the Tidewater section of Virginia continue, according to Peple.

AN HMO OFFERING dental care, Denticare of Virginia, established a beachhead in Richmond recently when it was offered to the 3,500 employees of the City of Richmond and signed up about 25% of them. For monthly premiums beginning at $8, Denticare's subscribers receive basic dental services from one of the dentists under contract with the Alexandria-based firm. About 20 Richmond dentists are participating in the plan.

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WARNINGS: Caution patients about activities requiring alertness (e.g., operating vehicles or machinery). Antihistamines are more likely to cause dizziness, sedation, and hypotension in elderly patients. Patients should also be warned about the possible additive effects of alcohol and other CNS depressants.

Usage in pregnancy: Safe use in pregnancy has not been established. Use only when the potential benefits have been weighed against the possible hazards to the mother and child. Note that an inhibitory effect on lactation may occur.

PRECAUTIONS: Use with caution in patients with a history of bronchial asthma, increased intraocular pressure, hyperthyroidism, cardiovascular disease, hypertension, hiatal hernia with reflux esophagitis, intestinal atony of the elderly or debilitated intestinal obstruction, myasthenia gravis, renal function impairment, and ulcerative colitis (severe).

Drug Interactions: MAO inhibitors, Alcohol or CNS depressants, especially anesthetics, barbiturates, and narcotics.

ADVERSE REACTIONS: Prolongs the response to nervous stimulation, potentiates the response to norepinephrine, and inhibits the response to tyramine.

Slight to moderate drowsiness occurs relatively infrequently with Chlorpheniramine Maleate. Other possible side effects common in antihistamines in general include perspiration, chills, dryness of mouth, nose and throat.

Cardiovascular System: Hypotension, headache, palpitations, tachycardia, extrasystoles.

Hematologic System: Hemolytic anemia, thrombocytopenia, agranulocytosis.

Nervous System: Sedation, dizziness, disturbed coordination, fatigue, confusion, restlessness, excitation, nervousness, tremor, irritability, insomnia, euphoria, paresthesias, blurred vision, diplopia, vertigo, tinnitus, acute labyrinthitis histeria, neuritis, convulsions.

Gastrointestinal System: Epigastric distress, anorexia, nausea, vomiting, diarrhea, constipation.

Genitourinary System: Urinary frequency, difficult urination, urinary retention, early menses.

Respiratory System: Thickening of bronchial secretions, tightness of chest and wheezing, nasal stuffiness.

DOSAGE AND ADMINISTRATION: Dosage should be individualized according to the needs and response of the patient. Adults: one capsule every 8 to 12 hours not to exceed 3 capsules daily. Not for use in children under 12 years of age.

OVERDOSAGE: Treatment of the signs and symptoms of overdosage is symptomatic and supportive. In the event of overdosage, emergency treatment should be started immediately.

Treatment: The patient should be induced to vomit, even if emesis has occured spontaneously. Vomiting by the administration of ipecac syrup is a preferred method. However, vomiting should not be induced in patients with impaired consciousness. Stimulants (analeptic agents) should not be used. Vasopressors may be used to treat hypotension. Short-acting barbiturates, diazepam or paraldehyde may be administered to control seizures. Hyperpyrexia, especially in children, may require treatment with tepid water sponge baths or a hypothermic blanket. Apnea is treated with ventilatory support.

HOW SUPPLIED: Green and clear capsules with green and white beads. Bottles of 100 tablets. NDC 0524-0031-01

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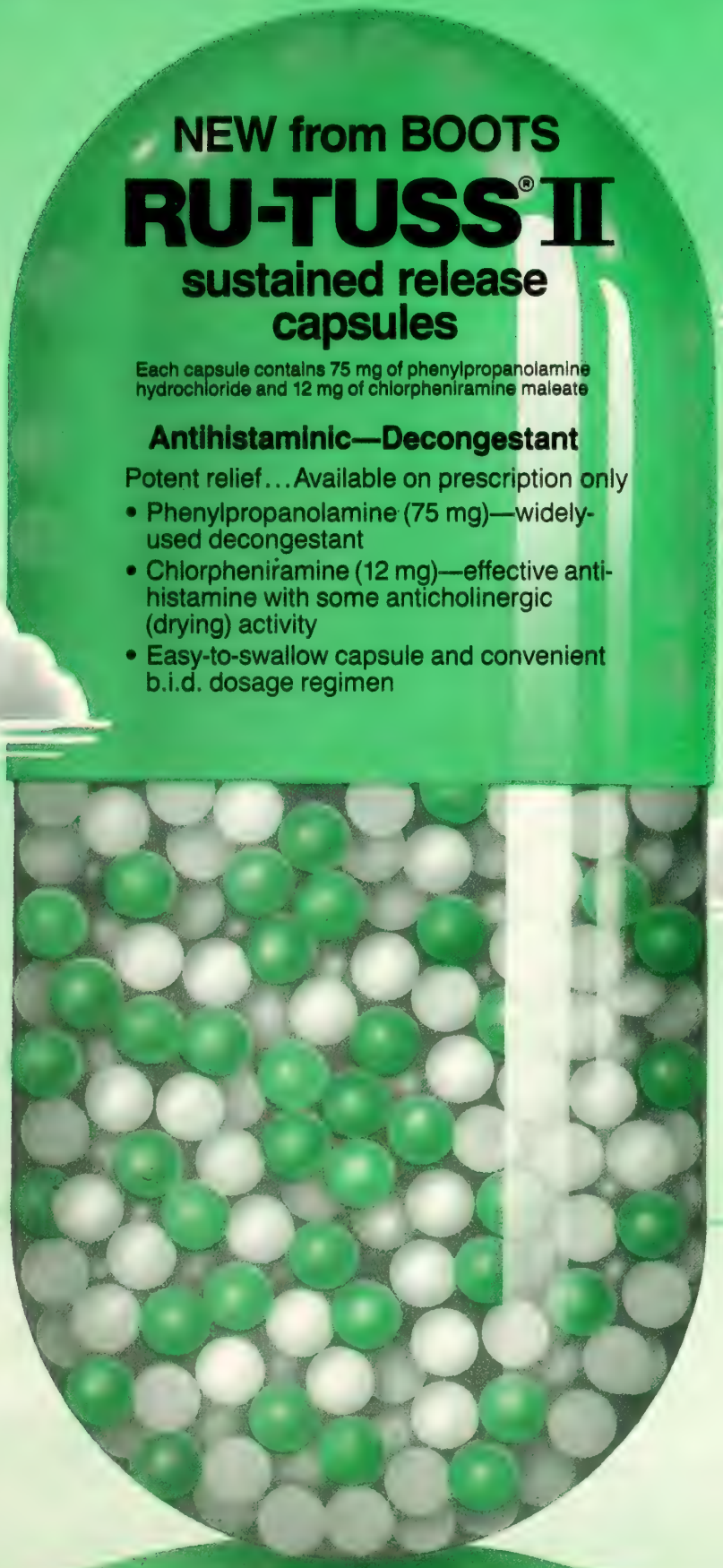

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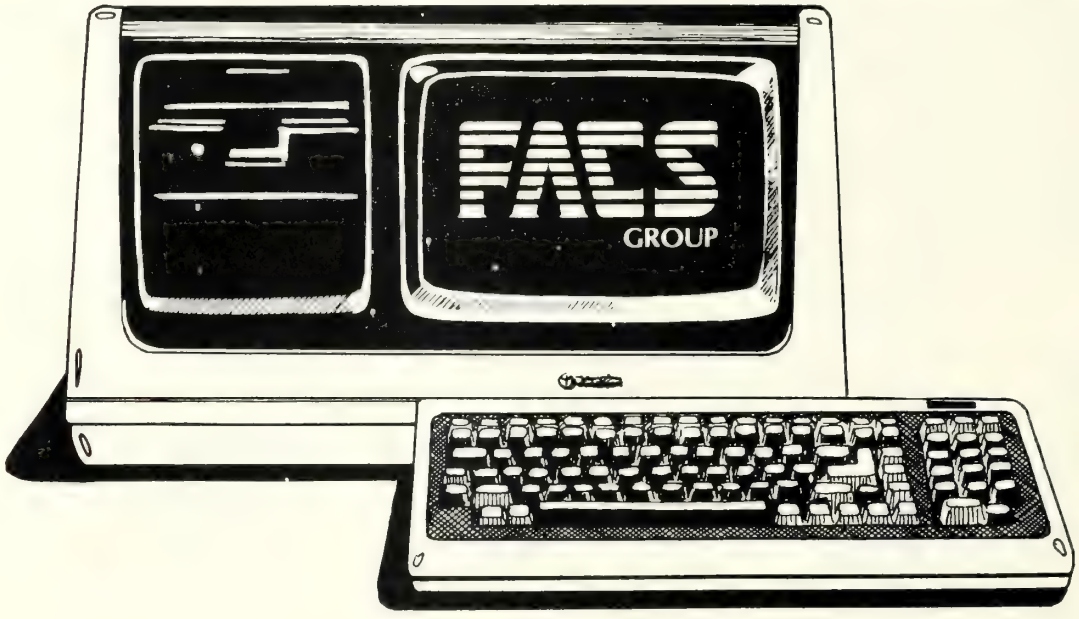

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MEETINGS

November 16
17th Annual William R. Whitman Memorial Lecture: What You Should Know About Immunology (Lewis-Gale Medical Foundation), *Salem*. Carolyn A. Goodykoontz, PO Box 44, Salem VA 24153, (703) 774-4022.

November 16-17
White House Symposium on Physical Fitness and Sports Medicine (President's Council on Physical Fitness and Sports), *Washington DC*. Fee: $175. 450 5th St., NW, Washington DC 20001, (202) 272-3424.

November 17
Annual Program on Cancer (Roanoke Memorial Hospital), *Roanoke*. Charles L. Crockett, Jr., MD, Roanoke Memorial Hospital, Roanoke VA 24033, (703) 981-7228.

November 18-19
Frontiers of Gynecologic Oncology (University of Maryland), *Baltimore*. Office of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

November 18-19
Progress in Pediatrics (Johns Hopkins University), *Baltimore*. 11 Cat. I credits. Fee: $80 for MDs; $40 for residents. Noreen Javornik, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-6046.

December 1-3
Comprehensive Breast Disease Symposium (Bowman Gray School of Medicine), *Winston-Salem, North Carolina*. 20 Cat. I credits. Fee $300. James F. Martin, MD, 300 S. Hawthorne Rd., Winston-Salem NC 27103, (919) 748-4505.

December 3
11th Annual Symposium on Geriatric Medicine (Johns Hopkins University), *Baltimore*. 5½ Cat. I credits. Fee: $40. Susan Bavaro, Office of CME, 720 Rutland Ave., Baltimore MD 21205 (301) 955-6046.

December 3
The Neck: Cervical Spondylosis (National Hospital for Orthopaedics and Rehabilitation), *Alexandria*. 8 Credit hrs. Irma Simpson, Medical Staff Coordinator, 2455 Army Navy Dr., Arlington VA 22206, (703) 553-2412.

December 4-6
Ross International Conference in Pediatrics, *Washington DC*. J. F. Lucey, MD, Medical Center Hospital of Vermont, Burlington VT 05401.

December 8-10
International Helen B. Taussig Symposium in Pediatric Cardiology (Johns Hopkins University/American Heart Association), *Baltimore*. Cat. I credits. Fee: $300. Carlita M. Kearney, Office of CME, 720 Rutland Ave., Baltimore MD 21205.

December 8-10
Pediatric Imaging Seminar (Bowman Gray School of Medicine), *Winston-Salem, North Carolina*. 20 Cat. I credits. Thomas E. Sumner, MD, 300 S. Hawthorne Rd., Winston-Salem NC 27103, (919) 748-4505.

December 10
Anxiety and Depression in Office Patients (Eastern State Hospital/Taylor Manor Hospital), *Williamsburg*. 6 Cat. I credits. Fee: $50. Mary Ann Ayd, PO Box 16315, Baltimore MD 21210, (301) 435-6562.

January 7-17
Medical Updates V: A Review of Recent Advances in Medicine (East Tennessee State University), *Vail, Colorado*. Sue Hutchinson, MPH, Office of CME, Johnson City TN 37614, (615) 928-6426, Ext. 204.

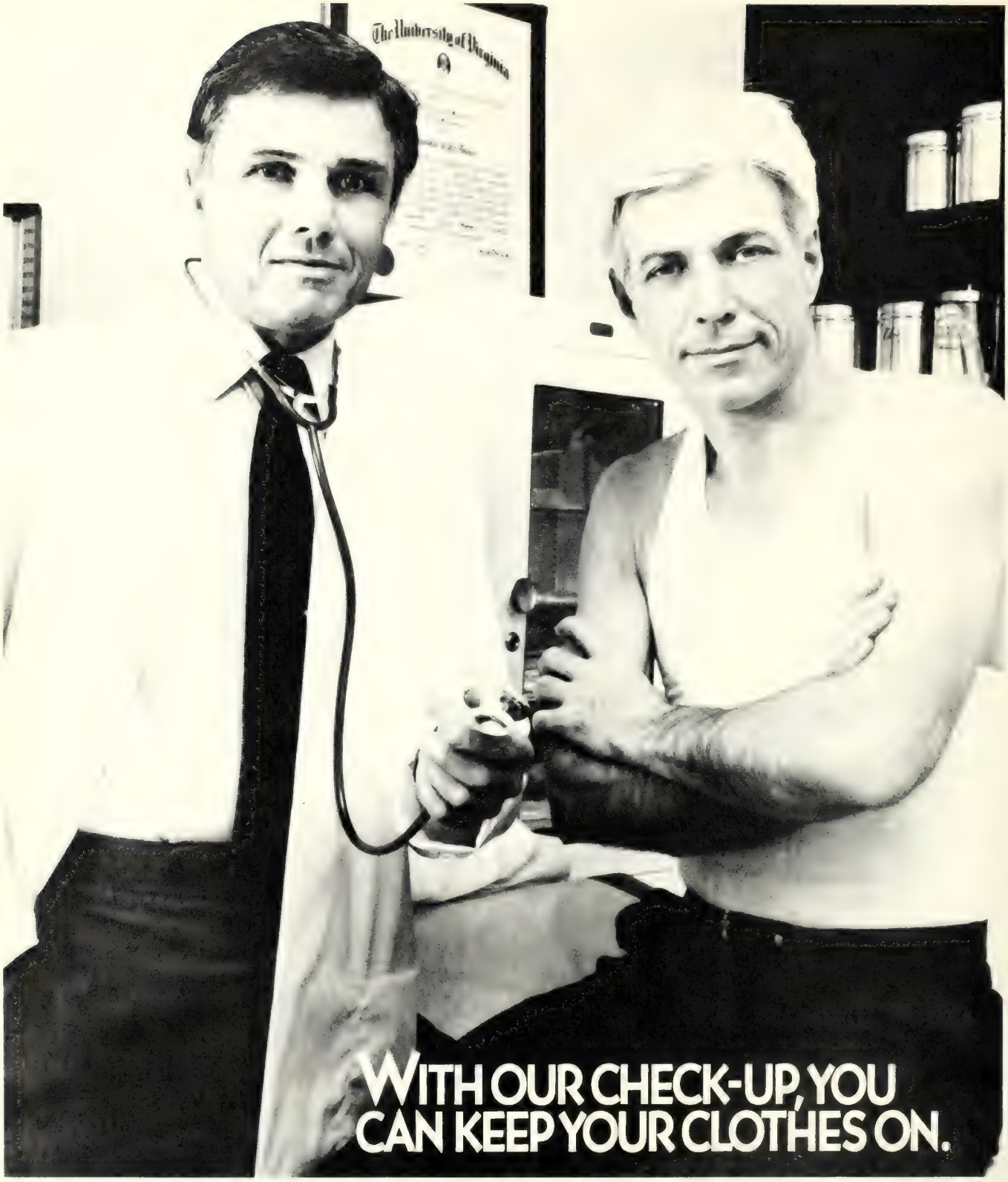
January 7
Regional Meeting, American College of Physicians, *Washington DC*. Wesley M. Oler, MD, 4800 Van Ness St., NW, Washington DC 20016.

January 19-21
Annual Meeting, Neurosurgical Society of the Virginias, *Williamsburg*. Robert P. Singer, MD, 1651 Parham Rd., Richmond VA 23229.

January 19
Controversies in the Diagnosis and Early Management of Breast and Colon Cancer (East Tennessee State University), *Johnson City, Tennessee*. Sue Hutchinson, MPH, Office of CME, Johnson City TN 37614, (615) 928-6426, Ext. 204.

January 29-February 3
Neuroimaging Update (University of Maryland), *Cancun, Mexico*. Program of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

January 30-February 2
The Alton D. Brashear Postgraduate Course in Head and Neck Anatomy (Department of Anatomy, Medical College of Virginia), *Richmond*. 40 credit hrs. Fee: $350, MDs; $200, residents. Hugo R. Seibel, PhD, Box 709, MCV Station, Richmond VA 23298.



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4. Is drinking affecting your reputation?

5. Have you ever felt remorse after drinking?

6. Have you gotten into financial difficulties as a result of drinking?

7. Do you turn to inferior companions and environment when drinking?

8. Does your drinking make you careless of your family's welfare?

9. Has your ambition decreased since drinking?

10. Do you crave a drink at a definite time daily?

11. Do you want a drink the next morning?

12. Does drinking cause you to have difficulty in sleeping?

13. Has your efficiency decreased since drinking?

14. Is drinking jeopardizing your job or business?

15. Do you drink to escape from worries or trouble?

16. Do you drink alone?

17. Have you ever had a complete loss of memory as a result of drinking?

18. Has your physician ever treated you because of your drinking?

19. Do you drink to build up your self-confidence?

20. Have you ever been to a hospital or institution due to drinking?

This questionnaire was originally developed at Baltimore's Johns Hopkins Hospital. "Yes" answers to three or more questions are a strong indication of alcoholism.

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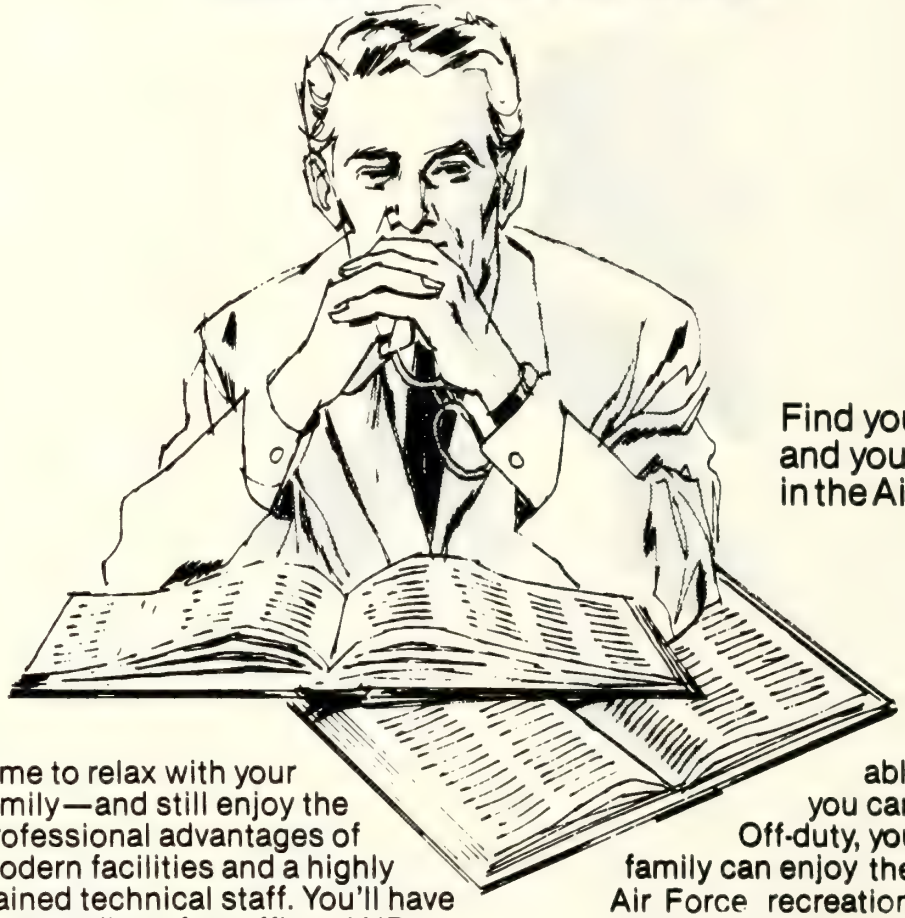

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cephalexin*	81% 343 339	85% 67 031	92% 67 027	16% 4398	80% 4365	22% 1834	13% 7571	12% 11 846
nitrofurantoin	97% 307 785	65% 61 162	7% 59 775	13% 3848	14% 3059	67% 1707	60% 7109	67% 10 830

*Analogous to cephalothin, the primary antibiotic disc used in testing
Source: The Bacteriologic Report, BAC-DATA Medical Information Systems, Inc., Winter Series 1981-82
Numbers under percentages refer to the projected number of isolates tested

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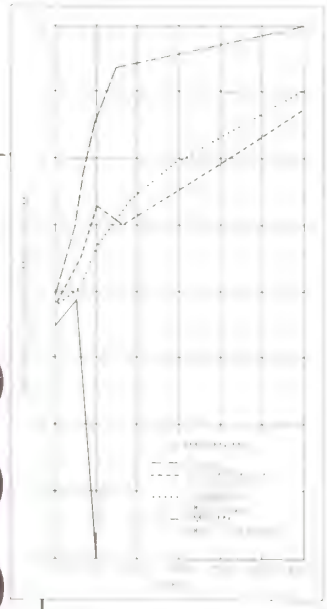

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Initial episodes of uncomplicated urinary infections should be treated with a single-agent antimicrobial.

Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

b.i.d. *for recurrent urinary tract infections*

**In vitro* data do not necessarily predict clinical results

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See next page for references and a summary of product information.

References: 1. Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Kramer MJ, Maunz YR, Robertson TL, Timmes MD: Morphological studies on the effect of subinhibitory and inhibitory doses of sulfamethoxazole-trimethoprim combination on *Escherichia coli*. Presented at the 12th International Congress of Chemotherapy, Florence, Italy, Jul 19-24, 1981. **3.** Spicehandler J *et al*: *Rev Infect Dis* 4:562-565, Mar-Apr 1982. **4.** Stamey TA: *Pathogenesis and Treatment of Urinary Tract Infections*. Baltimore, Williams & Wilkins, 1980, p. 13. **5.** Ronald AR: *Clin Ther* 3:176-189, Mar 1980. **6.** Cooper J, Brumfitt W, Hamilton-Miller JMT: *J Antimicrob Chemother* 6:231-239, 1980. **7.** Gower PE, Tasker PRW: *Br Med J* 1:684-686, Mar 20, 1976. **8.** Cosgrove MD, Morrow JW: *J Urol* 111:670-672, May 1974. **9.** Iravani A *et al*: *Antimicrob Agents Chemother* 19:598-604, Apr 1981. **10.** Schaeffer AJ, Flynn S, Jones J: *J Urol* 125:825-827, Jun 1981. **11.** Rous SN: *J Urol* 125:228-229, Feb 1981. **12.** BAC-DATA Medical Information Systems, Inc., Bacteriologic Reports, Winter Series, 1976-82

Bactrim™ DS

(trimethoprim and sulfamethoxazole/Roche)

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients. *Pregnancy:* Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, hepatocellular necrosis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: *Not recommended for infants less than two months of age.*

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS.

Usual adult dosage. 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets*, each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100 and 500, Tel-E-Dose* packages of 100. Prescription Paks of 20. *Tablets*, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500, Tel-E-Dose* packages of 100; Prescription Paks of 40. *Pediatric Suspension*, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml), cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension*, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per tea spoonful (5 ml), fruit-licorice flavored—bottles of 16 oz (1 pint).

ROCHE LABORATORIES
Division of Hoffmann-La Roche Inc
Nutley, New Jersey 07110

References

1. Stone PH, Turi ZG, Muller JE: Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104:672-681, September 1982.
2. Antman E, Muller J, Goldberg S, et al: Nifedipine therapy for coronary-artery spasm: Experience in 127 patients. *N Engl J Med* 302:1269-1273, June 5, 1980.

BRIEF SUMMARY

PROCARDIA* (nifedipine) **CAPSULES** For Oral Use

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents: (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy, Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72) and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

More detailed professional information available on request.

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"I can do things that I couldn't do for 3 yrs. including joining the human race again."

Quotes from an unsolicited letter received by Pfizer from an angina patient.
While this patient's experience is representative of many unsolicited comments received, not all patients will respond to Procardia nor will they all respond to the same degree.

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"My daily routine consisted of sitting in my chair trying to stay alive."

"My doctor switched me to PROCARDIA[*] *as soon as it became available. The change in my condition is remarkable."*

"I shop, cook and can plant flowers again."

"I have been able to do volunteer work...and feel needed and useful once again."

PROCARDIA can mean the return to a more normal life for your patients—having fewer anginal attacks,[1] taking fewer nitroglycerin tablets,[2] doing more, and being more productive once again

Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%)

for the varied faces of angina

PROCARDIA®
(NIFEDIPINE) *Capsules 10 mg*

*Procardia is indicated for the management of
1) Confirmed vasospastic angina.
2) Angina where the clinical presentation suggests a possible vasospastic component
3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

Please see PROCARDIA *brief summary on adjoining page*

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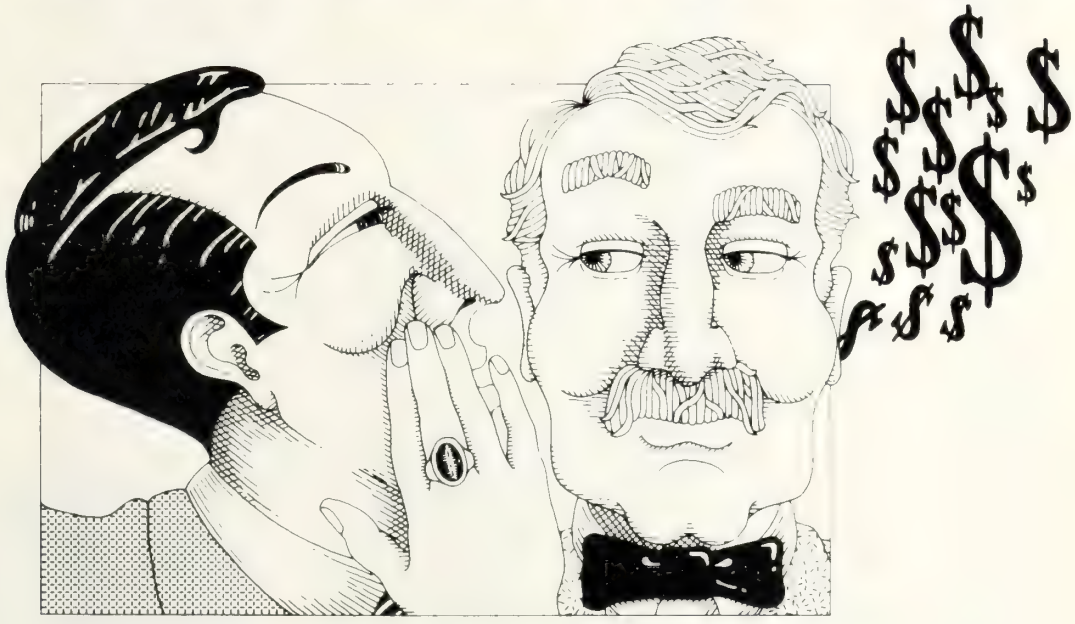

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Our psychoeducational treatment considers the needs of the total family

woman. Outpatient services provide support for a return to everyday life. An Employee Assistance Program provides business

A special program addresses the needs of chemically dependent women

management with skills in handling substance abuse problems that effect employee performance.
The Southwest Virginia Treatment Center is a Blue Cross Blue Shield service provider and is

Family Education is part of the total recovery program.

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The Southwest Virginia Treatment Center is fully licensed and accredited by the State of Virginia.

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And what is more reassuring to an excessively anxious patient than medication that promptly starts to relieve his discomforting symptoms? Valium® (diazepam/Roche) begins working within 30 to 90 minutes. Patients continue to improve in just a few days, and relief continues throughout the course of treatment.

There are other important benefits with Valium as well—along with its broad clinical range, Valium has an efficacy/safety profile that few, if any, drugs can match. This record has been achieved with extensive clinical experience, undoubtedly including yours. And, as you must have observed, side effects more serious than drowsiness, fatigue or ataxia rarely occur. Nevertheless, as with any CNS-acting agent, patients should be cautioned about driving, operating hazardous machinery or ingesting alcohol or other CNS-depressant drugs while taking Valium.

Yet another benefit Valium affords is flexibility. Available in 2-mg, 5-mg and 10-mg scored tablets, Valium enables you to titrate dosage to individual patient needs. For the geriatric patient, a starting dosage of 2 to 2½ mg once or twice a day is recommended. And, for patients who forget or skip medication, you can prescribe Valrelease™ (diazepam/Roche) 15-mg slow-release capsules, knowing that Valrelease will assure all the benefits of Valium 5 mg *t.i.d.* with the convenience of once-a-day dosage.

Discontinuation of Valium (or Valrelease) is typically as smooth as its start in short-term therapy. However, Valium and Valrelease should be discontinued gradually after more extended treatment. As you diminish dosage, the built-in tapering action of Valium and Valrelease will help avoid rapidly recurring anxiety symptoms and symptoms of withdrawal, and will help ease the patient's transition to independent coping when therapeutic goals have been achieved.

...that's one of the unique benefits of

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For a summary of product information, please turn the page.

ROCHE®

Valium® (diazepam/Roche) Ⓝ Tablets
Valrelease™ (diazepam/Roche) Ⓝ slow-release Capsules
Injectable Valium® (diazepam/Roche) Ⓝ

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, impending or acute delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in: relief of skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome. *Oral forms* may be used adjunctively in convulsive disorders, but not as sole therapy. *Injectable form* may also be used adjunctively in: status epilepticus; severe recurrent seizures; tetanus; anxiety, tension or acute stress reactions prior to endoscopic/surgical procedures; cardioversion

The effectiveness of diazepam in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient

Contraindications: Tablets or capsules in children under 6 months of age; known hypersensitivity; acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy

Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence

> ***Usage in Pregnancy*: Use of minor tranquilizers during first trimester should almost always be avoided because their use is rarely a matter of urgency and because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral forms adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures

INJECTABLE *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins, i.e., dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Injectable Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea, have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status. Not recommended for OB use

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of diazepam, *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or overse dation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed and tolerated)

The clearance of diazepam and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear

INJECTABLE Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, observed in patients during and after diazepam therapy are of no known significance.

INJECTABLE Venous thrombosis/phlebitis at injection site, hypoactivity, syncope, bradycardia, cardiovascular collapse, nystagmus, urticaria, hiccups, neutropenia

In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported

Dosage: Individualize for maximum beneficial effect

ORAL *Adults*: Anxiety disorders, relief of symptoms of anxiety—Valium (diazepam/Roche) tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 Valrelease capsules (15 to 30 mg) daily. Acute alcohol withdrawal—tablets, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; or 2 capsules (30 mg) the first 24 hours, then 1 capsule (15 mg) daily as needed. Adjunctively in skeletal muscle spasm—tablets, 2 to 10 mg t.i.d. or q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily. Adjunctively in convulsive disorders—tablets, 2 to 10 mg b.i.d. to q.i.d.; or 1 or 2 capsules (15 to 30 mg) once daily.

Geriatric or debilitated patients: Tablets—2 to 2½ mg 1 or 2 times daily initially, increasing as needed and tolerated (see Precautions). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose

Children: Tablets—1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use in children under 6 months). Capsules—1 capsule (15 mg) daily when 5 mg oral Valium has been determined as the optimal daily dose (not for use in children under 6 months)

INJECTABLE Usual initial dose in older children and adults is 2 to 20 mg I.M. or I.V., depending on indication and severity. Larger doses may be required in some conditions (tetanus). In acute conditions injection may be repeated within 1 hour, although interval of 3 to 4 hours is usually satisfactory. Lower doses (usually 2 to 5 mg) with slow dosage increase for elderly or debilitated patients and when sedative drugs are added. (See Warnings and Adverse Reactions.)

For dosages in infants and children see below; have resuscitative facilities available

I.M. use: by deep injection into the muscle

I.V. use: inject slowly, take at least one minute for each 5 mg (1 ml) given. Do not use small veins, i.e., dorsum of hand or wrist. Use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion

Moderate anxiety disorders and symptoms of anxiety, 2 to 5 mg I.M. or I.V., and severe anxiety disorders and symptoms of anxiety, 5 to 10 mg I.M. or I.V., repeat in 3 to 4 hours if necessary; acute alcohol withdrawal, 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary. Muscle spasm, in adults, 5 to 10 mg I.M. or I.V. initially, then 5 to 10 mg in 3 to 4 hours if necessary (tetanus may require larger doses); in children *administer I.V. slowly*; for tetanus in infants over 30 days of age, 1 to 2 mg I.M. or I.V., repeat every 3 to 4 hours if necessary; in children 5 years or older, 5 to 10 mg repeated every 3 to 4 hours as needed Respiratory assistance should be available

Status epilepticus, severe recurrent convulsive seizures (I.V. route preferred), 5 to 10 mg adult dose administered slowly, repeat at 10- to 15-minute intervals up to 30 mg maximum. Repeat in 2 to 4 hours if necessary, keeping in mind possibility of residual active metabolites. Use caution in presence of chronic lung disease or unstable cardiovascular status. Infants (over 30 days) and children (under 5 years), 0.2 to 0.5 mg slowly every 2 to 5 min., up to 5 mg (I.V. preferred). Children 5 years plus, 1 mg every 2 to 5 min., up to 10 mg (slow I.V. preferred); repeat in 2 to 4 hours if needed. EEG monitoring may be helpful

In endoscopic procedures, titrate I.V. dosage to desired sedative response, generally 10 mg or less but up to 20 mg (if narcotics are omitted) immediately prior to procedure; if I.V. cannot be used, 5 to 10 mg I.M. approximately 30 minutes prior to procedure. As preoperative medication, 10 mg I.M.; in cardioversion, 5 to 15 mg I.V. within 5 to 10 minutes prior to procedure. Once acute symptomatology has been properly controlled with injectable form, patient may be placed on oral form if further treatment is required

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CON

By Ann Gray,
Executive Editor

Gone forever, no doubt, are the days when doctors ran their own hospitals, but two groups of Virginia physicians are recapturing some of that lost entrepreneurial ground by launching their own freestanding ambulatory surgery centers (ASCs).

Pioneering the two facilities are an association of seven Richmond urologists headed by Dr. C. M. Kinloch Nelson and the sixty-six physicians who own the multispecialty Lewis-Gale Clinic in Roanoke.

Freestanding ambulatory surgery centers exemplify the new wave of outpatient service designed to bypass the high cost of hospitalization. They accommodate "in-and-out" surgery, which is a cut above office treatment but not serious enough to necessitate hospital admission. Many Virginia hospitals have installed ASCs in the recent past. The two versions,

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physician-owned and hospital-based, are competitive.

Whether in a hospital or a physicians' office building, these ambulatory surgery centers are clearly demarcated as separate entities; hence the word "freestanding." They are subject to full-dress regulations by the State, and to establish one, you have to get a certificate of public need (CON).

In Roanoke, the Lewis-Gale Clinic's application to start a physician-owned ASC was shepherded through the long and litigious certificate of need course by the administrator, Darrell D. Whitt. He could have turned it over to legal counsel, but that would have cost a chunk of money, and "I enjoyed doing it," he says. His petition for the ASC "went right through without a hitch," in Whitt's words.

It is not uncommon for professional administrators to run their applications over the CON hurdles, but physicians are rarely seen on this track. Yet Dr. Nelson singlehandedly sought and won approval of the ambulatory surgery center his group of urologists is to operate in Richmond. He, too, thought of hiring legal talent for the task, he admits, but the lawyer "wanted $10,000 for starters. *For starters*," he repeats incredulously. And what did it cost Dr. Nelson? He shrugs. "$25. For parking fees and photocopies."

Like Whitt, Dr. Nelson had a working familiarity with the certificate of need system but on the other side of the fence: He has served for four years as The Medical Society of Virginia's representative to the Capital Area Health Advisory Council (CAHAC), and the Council is one of the reviewing agencies for certificates of need in Richmond. His Council membership conferred no immunity from the rigors of the application protocol, however; Dr. Nelson had to maneuver through every jot and tittle of it.

Unlike Whitt, he had a rough time of it. In fact, when his application finally wound up in the office of the Commissioner of Health, it was denied. Only by appealing the decision and winning reversal did Dr. Nelson get his certificate of need.

Here is the story of his adventures.

THE Health Care Financing Administration, which is part of the Department of Health and Human Services, is very keen on freestanding ambulatory surgery centers as a means of lowering Medicare costs and recently initiated a cost-free reimbursement plan for ASCs. It was Dr. Nelson's introduction to this new program that propelled him into the certificate of need process. He recalled the circumstances in an interview that took place while his petition was still pending.

"We have a lot of Medicare patients, and I went down to the Medicare office to find out about hooking up with their computer. That was last December, when I was working out some things with the architect." He was referring to the renovation of the building he and his associates bought in November 1982; it had previously been the headquarters of the Richmond Board of Realtors.

"The man I was talking to at Medicare said, 'I wonder if you have any ideas about how we can implement this,' and he showed me an issue of the *Federal Register* with a lot of information put together by the Health Care Financing Administration about a new prospective payment plan for Medicare patients in ambulatory surgery centers. It even gave the names and phone numbers of four people at the HCFA you could call and talk to about it."

From his briefcase Dr. Nelson extracted a sheaf of papers densely covered with small type. They were photocopies of pages 34082-34101 of the *Federal Register* for August 5, 1982. The title read "Medicare Program: Ambulatory Surgical Services." There followed column after column of rules, regulations, standards and instructions.

Bringing up the rear was a list of covered procedures with payment amounts. The procedures were cat-

egorized according to system, i.e., respiratory, nervous, digestive, musculoskeletal and so on, and each category was divided into four payment groups. Thus for a cystourethroscopy with retrograde pyelogram, the program offered $231. In contrast, the urologist said, at a Richmond hospital whose charges were neither highest nor lowest but somewhere in between, the charge for this procedure was $460. "That's comparing apples and apples," he emphasized. "Same procedure, same quality."

And these listed payments are the facility fees one conventionally associates with hospitals, correct? asked the interviewer. Correct, said Dr. Nelson. But in this case they would go to the urologists' ambulatory surgery center, and the physicians fees would also be billed as usual, correct? "Sure," Dr. Nelson answered swiftly, "we stand to make more money, but I'm not hiding that and I'm not ashamed of it." Although, he added, his face clouding over, some people seemed to find something wrong with it.

What urologic procedures would you perform in an ASC that could not be done in an office? the interviewer inquired.

"About 40% of our cystoscopies with retrograde pyelograms are now performed in the hospital, because the patients need sedation," Dr. Nelson replied. "We'd do those in the ambulatory surgery center. Some urologists perform vasectomies and circumsions in the office, but we don't think the office is safe enough for those; the ambulatory surgery center would be, however. We're now doing prostate and bladder biopsies in the hospital, too, partly for sedation, partly for after-care; those could be done in the ASC.

"We specifically would not want to shift office treatment into the ambulatory surgery center," he asserted, "and we'll have rigid internal controls to see that that is not done."

LIKE EVERYONE ELSE who undertakes the certificate of need course, Dr. Nelson began with a little warm-up protocol. For the application form he got in touch with Mrs. Marilyn West, director of the Division of Resources Development at the Virginia Department of Health. Once he'd mastered the form's marathon 19 pages, he took both the application and himself to the director of the nearest health systems agency, who was Michael Osorio of the Central Virginia HSA. Osorio pronounced the petition fit. Back it went to West. When she accepted it for review, it became Osorio's task to set up three hearings.

All three of these hearings would be open to the public and duly advertised in the local newspapers, but the first one is the designated public hearing. It amounts customarily to a small, friendly rehearsal for the petitioner's future appearances before two reviewing bodies, and such was the case with Dr. Nelson's public hearing on March 24.

Some questions were asked by Larry D. Jones, a provider-member of CAHAC and its chairman, who would not be able to attend the coming review by that group. Jones is a senior marketing representative for Pru-Care, a Richmond health maintenance organization.

Would the proposed office-located ambulatory surgery center so shift patients away from hospitals that it would lead to an increase in hospital costs? he asked. Dr. Nelson responded that the effect on hospitals would be insignificant, that prospective payment was going to change the costs picture in any event.

How did the proposed freestanding ASC fit into such developments as "emergicenters"? Ambulatory surgery centers are part of the same big trend to lower-cost settings for patient care, was the response.

What ancillary services would be available? Jones continued. X-ray and renal scan, Dr. Nelson said, and of course the requisite pathology lab.

Support for Dr. Nelson's application was offered by John N. Simpson, president of Richmond Memorial Hospital. The proposed ambulatory surgery center might well be in competition with his hospital, Simpson said, but if the urologists could provide less costly treatment, then they should be allowed to do so.

Hearing adjourned. Dr. Nelson's application would go forward.

More precisely, it would go upward, for the road to a certificate of need ascends through the local health advisory council, the regional health systems agency, and the Statewide Health Coordinating Council (SHCC) to the top, where sits Dr. James B. Kenley, Virginia's Commissioner of Health. For Dr. Nelson, the first stop would be Planning District 15's review body, the very Capital Area Health Advisory Council of which he is a member.

AS DOES ITS COUNTERPARTS in each of Virginia's 21 other planning districts, the Capital Area Health Advisory Council has 30 members, of whom 16 are consumers, 14 are providers. All serve without honoraria. Eight of the consumer-members are appointed by the counties within the planning district, and eight are elected by the Council.

Of the provider-members, 12 are appointed by provider organizations. In PD 15, these organizations are The Medical Society of Virginia; Medical College of Virginia; South Central PSRO; Central Vir-

ginia Hospital Council; Virginia Nurses Association; Blue Cross Blue Shield of Virginia; Virginia Health Care Association (long-term care facilities); Virginia Association of Allied Health Professions; Neuropsychiatric Society of Virginia; and Region IV Consortium Mental Health/Mental Retardation/Substance Abuse. The other two provider-members are elected by the Council.

Ten days before each of its monthly meetings, members of the Council receive from the staff an analysis of each application to be considered at the coming session, with the staff's recommendations as to the application's disposition. On this staff report, Dr. Nelson's proposed ASC stubbed its toe.

Certain aspects of the application were "commendable," the report admitted, but there were other considerations. The regional Health Systems Plan clearly states, the staff noted, that free-standing ambulatory services should be established only when hospital-based surgical suites in the planning district equal or exceed 80% of the Plan's stated minimum utilization figure of 1,000 operations per room per year. A 1981 survey of the 11 hospitals in PD 15, however, yielded an average usage rate of 768.51 procedures per OR per year, well below the 80% specification.

Moreover, the contention that the proposed ASC would be more cost-effective was "probably a moot one," in the staff's opinion, because prospective payment was coming to hospitals, too, and there were four hospital-based ASCs with dedicated operating rooms already doing business in the planning district, and another was under construction.

The staff expressed grave concerns about shifts that could rock the health-care boat—shifts of costs in hospitals due to loss of revenue, shifts of patients from office treatment to ambulatory surgery, also cost-inflationary.

In summary, the staff recommended denial of Dr. Nelson's application for a certificate of need.

COPIES OF THE STAFF report had been sent to CAHAC members before they convened on March 28. Dr. Nelson was there as both Council member and applicant. When the agenda proceeded to consideration of his application, he was asked to describe the project for which he was requesting a certificate of public need.

Available to him nearby was a podium, flip chart, audio-visual equipment and overhead projectors. Many CON applicants need these props for the copious graphic material with which they buttress their pleas. Dr. Nelson's material, however, was "all in his head," as one observer recalls, and he stayed seated at the table and talked from there.

He practiced urology with six other physicians, Dr. Nelson began: Austin I. Dodson, Jr.; J. Edward Hill; G. Bryan Duck; Frank N. Pole; William H. Atwill; and Gary B. Bokinsky. They wanted to install in their office building an ambulatory surgery center with two operating rooms, plus X-ray and renal scan. Their lab was entirely adequate to meet pathology needs. The facility would have a staff of four, led by a registered nurse. There would be appropriate medical review. No capital expenditures were required; the urologists had recently bought an office building, it had been renovated to suit the doctor's office practice, the ASC installation would be part of the renovation costs.

He emphasized the convenience to patients of the proposed ASC and wound up with a description of the HCFA's prospective payment program for Medicare patients. He had sent all details of the proposed surgical center to the HCFA people in Washington, and they had responded that all systems were go.

The acting chairman, David W. Pentico, Richmond statistician and consumer-member of the Council, entertained questions.

What about safety precautions for patients? one Council member asked. The urologists had set up an agreement with Richmond Memorial Hospital for backup in case of emergency, Dr. Nelson answered.

Was it to be closed staffing? No, Dr. Nelson replied, it would be open staffing; there are 16 other urologists in the area who are not associated with hospitals, and they would be free to use the center.

Would the Medicare program cover charges for X-ray and renal scan? No, those would not be covered, the urologist explained, but they would be charged at rates lower than those of existing facilities. How would they be billed? Separately.

There were a few other questions, then a vote was solicited. Dr. Nelson abstained. The 14 other members present voted unanimously to approve the application because 1) "the proposal appears to be cost-effective" and 2) "the free-standing ambulatory surgery center offers the provision of alternative medical care."

Next hurdle: The board of directors of the Central Virginia Health Systems Agency.

LIKE THE SUBSIDIARY health advisory councils, the boards of each of Virginia's five HSAs are comprised of 16 providers and 14 consumers. They are appointed and elected in much the same way, except that HSA directors are drawn from the region rather than the locality; thus consumer-directors are appointed not by counties but by the planning

Footprints on the sands of certified need

REMEMBER THE TUG OF WAR five years ago when the prototype CAT scanner tangled with Virginia's certificate of public need process? The health planners, from whom all permits flow, were alarmed by the new machine's price tag; those applying for the technology contended that it would save money in the long run. The providers prevailed, and scanning became a routine diagnostic procedure.

Today, applications to buy the second generation of CATs are proceeding quietly through the certificate of need system, accompanied by a growing market in second-hand scanners. Controversy now centers on higher tech at even higher cost, nuclear magnetic resonance.

Thus is history written in the certificate of public need program. At the program's outset, ten years ago, most of the applications were for bed-intensive projects. Today, the addition of hospital beds is frowned on in most localities, and nursing home beds reached such a peak of proliferation recently that a moratorium was placed on their construction. When the moratorium was lifted in July, a few nursing homes made haste to apply for new beds, but it is not expected that the reviewers will make haste to grant them.

Most certificates of need today have to do with new equipment and new service. In the equipment category, two technologies are challenging the CAT scanner activity: laser-ophthalmology, for which five Virginia hospitals were given CONs in the year ending July 1983, and digital vascular imaging, approved for six hospitals in the same period.

The costliest equipment yet is nuclear magnetic resonance. Richmond's Chippenham Hospital applied to spend $3.5 million to install Virginia's first NMR scanner, but the application was denied; until its clinical usefulness is known, said the Commissioner of Health, these new scanners should be placed in research-teaching hospitals. We'll appeal, retorted Chippenham officials.

As for new services, home health care is at flood tide. Of the 245 requests for CON application forms in the year ending July, 68 were for home health projects. A lot of these suppliants lost heart when they saw the form, a grueling document 19 pages long. Many who perservered were denied. Ten were granted certificates—five commercial firms, four hospitals and one clinic. The expansion of three existing home health agencies was also approved.

In the same 12-month period a flurry of hospitals asked to install cardiac catheterization laboratories, this rush occurring when the CC labs already in business began exceeding the official highwater usage mark of 500 procedures per year. Six new CC labs were approved and two hospitals were given permission to add second ones. Whereupon the planners allowed as how they'd better damp down this enthusiasm by raising the usage quotient.

Definitely a comer in the new services category is hospice care. In the same 12-month period, five new hospice programs were granted certificates of public need in Virginia: three independent operations, one hospital-based, one in a retirement complex. A surge of hospice activity is expected when the new Medicare reimbursement program for hospice care gets into gear.

Ambulatory surgery centers have been touted as a new medical service with big growth potential, and the 12-month record shows receipt at the Department of Health of more than a dozen letters of intent to apply for freestanding ASCs. Some of these letters have come from individual physicians, and since corporate entities overwhelmingly dominate certificate of need records, their names surprise the reader—a vascular surgeon in Virginia Beach; two general practitioners, one in Bristol, one in Norfolk; a plastic surgeon in Richmond; an ophthalmologist in Danville.

But as this was written only two certificates of need had been granted for physician-owned ambulatory surgery centers. The story is told in these pages. A.G.

districts of the region. The directors are volunteers; there is no honorarium.

The meetings of both council and board are similar, too. The CON petitioner appears to answer questions. The directors receive for study the same staff report that was submitted to the council.

On April 6, 1983, the board of directors of the Central Virginia Health Systems Agency met in the Blue Cross Blue Shield building in Richmond. Introducing Dr. Nelson's application for discussion, Mrs. W. Hamilton Crockford III, consumer board member and its chairwoman, noted that the health advisory council had forwarded its recommendation for approval of the application.

But the staff's report recommended denial, a board member observed to Jack V. Quigley, HSA health planning specialist. What aspects of the application recommended approval?

The proposal to provide outpatient surgery procedures, the staff man returned, is definitely in the direction of cost-effective delivery of health care.

Have any hospitals raised any objections to your proposal? No, he'd had no objections from hospitals, Dr. Nelson related, and from one hospital had come a positive response.

Would Dr. Nelson comment on the relative costs? He cited the $460 hospital charge versus the $231 of the Medicare fee schedule.

One person couldn't understand about the absence of capital expenditures. Dr. Nelson assured the questioner that installation costs would be subsumed by the renovation budget.

"I'm in favor of the application myself," volunteered James L. Gore, vice president of provider services for BLue Cross Blue Shield of Virginia. "Sure, it may hurt hospitals for a while, but I think it's a legitimate form of competition, and unless we allow some legitimate competition in this area, we're not going to answer all the questions regarding the cost of health care."

Dr. Wyatt S. Beazley III, Richmond surgeon who is one of The Medical Society of Virginia's representatives to the Central Virginia HSA board, joined Gore's affirmative position.

"We've been searching for ways to save money," Dr. Beazley said, "and this is a new way to approach things. My concern is why you even have to have a certificate of need for it, because every doctor who wants to do something like this is going to have to apply. The quality of service is going to be just as good as it is in the hospital, I think, and the people who are going to benefit are the patients."

Whereupon there was a motion to approve for the reasons of 1) cost containment and 2) convenience to the public. The motion carried by a roll-call vote of 15 ayes and two nays.

Onward and upward to SHCC.

THE NATIONAL NETWORK of health systems agencies sprung up in response to legislation passed by Congress in 1974 and calling for oversight of certain aspects of American medical care, including accessibility, quality and costs. Each regional HSA is a non-profit, non-stock corporation funded by the United States Treasury.

The State of Virginia contributes to this apparatus through the Statewide Coordinating Council, which is a creature of the State and is funded equally by the State and the federal government.

Virginia's governor appoints the 31 members of the Statewide Council—two consumers and two providers from each of the five HSA regions and 11 members appointed at large. Consumers dominate the at-large appointments; the ratio at present is eight consumers to three providers. Virginia's physicians are always represented on the Statewide Council. Dr. George E. Broman, Culpeper, was SHCC chairman for the recent 1979-1981 term.

Each SHCC member must belong to one of two committees. The Planning and Policy Analysis Committee works on the State Health Plan and the State Medical Facilities Plan; in these two plans are contained the basic criteria and standards for medical care in Virginia and projections for future medical needs. The Health Facilities and Services Review Committee considers certificates of need.

The Review Committee meets once a month, usually in Richmond but sometimes in other cities of the state. Its members are paid $50 per diem and travel expenses. Before each meeting, each committee member receives a dossier on every CON petition to be considered at the coming session; this includes information on all that has gone before in the review process and recommendations from the Division of Resources Development for disposition of each application.

The state staff's appraisal of Dr. Nelson's application sang the same song, second verse of the earlier HSA staff report. According to current standards in Virginia's two Plans, "there is no need for additional surgical facilities in PD 15," nor has the applicant "demonstrated that the need for surgical services cannot be accommodated by existing facilities." The proposal "could increase health systems costs" through the same cost-shifting that had worried the HSA staff, the report continued, and although ambulatory surgery can be considered "a less costly and more efficient alternative to inpatient surgery, prospective reimbursement

Kenley, study group see need for change

Both Virginia's Commissioner of Health and a legislative study group appointed by the 1983 General Assembly believe the certificate of need process needs changing.

"It's time, I think, to debate and reexamine certificate of need," Dr. James B. Kenley told VIRGINIA MEDICAL's reporter. "I feel some radical changes might be made in it, and right now I'm working on some ideas as to what those changes might be. Of course, any changes must come from the General Assembly, not this office. I can only suggest."

Dr. Kenley was not ready to be specific when he was interviewed, but the study group's ideas for change are reported to include exempting home health services from the CON process; appointing two consumers to share decision-making powers with Dr. Kenley; and reducing the seven-stage application procedure described in the accompanying article to four steps: public hearing, HSA board review, decision, one appeal. After a recent meeting, the group asked legal counsel to draft tentative legislation aimed at these changes, which fit some of the Commissioner's concerns.

"We've got to come up with some efficiencies," he said. "We're spending too much time and money on it, and we don't have the personnel to cover all the aspects as it's set up.

"And it amounts to a quote 'franchise', let's face it," he added, "with a lot of dollars involved and a whole host of problems legally and competitively."

In the first six months of 1983, 86 applications for certificates of need passed through the review process and wound up on Dr. Kenley's desk for disposition; they represented proposed capital expenditures totalling $115 million. Of these petitions, 60% were approved, but the approved projects signified in dollars only 21% ($33 million) of the total amount reviewed. A.G.

should encourage more effective utilization of existing facilities."

The state staff recommended denial.

THE SHCC REVIEW COMMITTEE met on April 20 to consider five applications for certificates of public need—for a home health agency, a rehabilitation service, the purchase of a used CAT head scanner, more beds for a psychiatric hospital, and Dr. Nelson's freestanding ambulatory surgery center.

First the committee turned down the petition for the home health agency. Then it gave its attention to the urologists' project. The acting chairman, Carroll I. Leith, Jr., Alexandria, asked Dr. Nelson to outline his proposed ASC. Leith, a consumer-member of SHCC, is a retired federal employee.

Dr. Nelson complied, speaking extemporaneously and citing the support for the project of the Health Care Finance Administration, Richmond Memorial Hospital and Blue Cross Blue Shield of Virginia.

Then HSA staff man Quigley reported that the board of directors of the Central Virginia Health Systems Agency had unanimously approved Dr. Nelson's application because the proposed facility was viewed as an effective approach to cost containment.

Next, as the minutes of the meeting relate, came the questions.

To Dr. Nelson: How many other urologists are associated with this project? Dr. Nelson went into his opening-staffing routine.

To Quigley: Why isn't the St. Mary's ambulatory surgery center in operation yet?

This questioner was recalling the committee's recent approval of an application for installation of an ASC at St. Mary's Hospital in Richmond; the new service was then being built on the hospital's seventh floor at a cost of almost $2 million. St. Mary's Hospital and Dr. Nelson's office building are both in Planning District 15. They are, in fact, only a few blocks away from each other.

Quigley replied that the St. Mary's facility was still under construction and staff was being searched for it.

"It's my feeling," commented a committee member, "that if this project goes into an area where the same services are offered, it will increase costs. We must be concerned about the existing facilities, and not approve a service that would shift patients away from them."

The HSA board did not feel, Quigley countered, that Dr. Nelson's facility would cause anyone to raise costs, and Dr. Nelson himself inserted that a facility of the type he was proposing might even encourage hospitals to lower their costs.

Do the urologists have any relationship to the medical school? another committee member wanted to know, referring to the Medical College of Virginia. Only in that

three of them are on MCV's clinical staff, the urologist replied.

Another committee member observed that the committee seemed to be overlooking the fact that the State Medical Facilities Plan has standards and guidelines that must be adhered to, and the state staff had pointed to existing surgical capacity in PD 15.

So saying, he moved to recommend denial of the application for the reasons given by the staff. The motion was promptly seconded and passed on a vote of 7 to 5. Exit Dr. Nelson, in pain.

"It was a terrible meeting," the urologist recalls. "I knew I was in trouble. They were so. . . adversarial."

Quigley does not remember the Review Committee as adversarial. There were a lot of new SHCC members at that hearing, he notes. They were fresh from studying the State Plan and were taking their duties very seriously.

In any event, the committee at that hearing refused all five CON applications set before them, but the company of these fellow rejects was small comfort to the urologist, who could only pin his hopes on a reprieve from the final arbiter, Commissioner Kenley.

The word from Dr. Kenley arrived in Dr. Nelson's mail two weeks after the SHCC hearing.

After "considering all factors," the Commissioner wrote, he was denying the application because 1) no more operating rooms, either outpatient or inpatient, were needed to accommodate the patient population in the area in question; 2) the State Plan says that no operating rooms should be added in an area until the existing ORs average 1000-1400 per year; and 3) the anticipated lower costs of the proposed freestanding ASC would not offset the costs generated by creating unneeded surgical capacity.

Dr. Nelson was entitled to appeal the decision if he wanted to, Dr. Kenley added.

DR. NELSON definitely wanted to and duly showed up for the appeal on June 21 at the James Madison Building, home of the Department of Health in Virginia's capital city. Appointed by Dr. Kenley to conduct the appeal was Dr. Bedford E. Berrey, Assistant State Health Commissioner. Mrs. West and an assistant were on hand as staff. With Dr. Nelson came Dr. Richardson Grinnan, vice president for medical affairs, Blue Cross Blue Shield of Virginia, who was there to pledge the insurance carrier's support for the urologists' surgical center.

Two appeals, one informal, one formal, are open to an applicant whose request for a certificate of public need has been denied. This was the informal appeal. The three men and two women talked pleasantly for about an hour and a half, but the truth was that Dr. Nelson had his dander up, as revealed by the typewritten plea he left with Dr. Berrey.

First he took aim at the assertion that there was enough surgical capacity in PD 15 to accommodate the available patients. He cited the Maryland Health Plan, which says that when a hospital begins operating at 80% or higher efficiency, scheduling difficulties become severe, in-house procedures are delayed, and elective surgical admissions are postponed or cancelled. All the add costs to the system and also increase patient anxiety, destroy work plans, and leave families and baby-sitters hanging.

The Commissioner's assertion that his project would not save the system money was Dr. Nelson's next target.

"This is pure and total conjecture by the Commissioner," he

Providing the physician's point of view

Four Medical Society of Virginia members are serving on the Statewide Health Coordinating Council: Mary Tom Long, MD, Roanoke; William W. Martin, Jr., MD, Richmond; John A. Rawls, MD, Richmond; and Malcolm Tenney, Jr., MD, Staunton.

And these MSV members contribute their services to the boards of directors of Virginia's five health systems agencies:

- Region I, Northwestern Virginia HSA: Richard M. Senfield, MD, Harrisonburg.
- Region II, HSA of Northern Virginia: John F. Heath, MD, McLean; Karl H. R. Hellinger, MD, Manassas; and Herbert S. Hopwood, MD, Arlington.
- Region III, Southwest Virginia HSA: Robert F. Baxter, MD, Grundy; John D. French, MD, Martinsville; and Mary Tom Long, MD, Roanoke.
- Region IV, Central Virginia HSA: Wyatt S. Beazley III, MD, Richmond; Charles W. Coppedge, MD, Farmville; Warren W. Koontz, Jr., MD, Richmond; and Frederick S. Vines, MD, Richmond.
- Region V, Eastern Virginia HSA: Joseph G. Fiveash, Jr., MD, Norfolk; Sidney S. Loxley, MD, Chesapeake; Jess P. Miller, MD, Hampton; and John W. Theimeyer, Jr., MD, Norfolk.

said, "and is erroneous in this new era of competition and prospective payment." Who are the parties most concerned with the system's cost? the urologist's plea asked. "Those that pay for it directly; in our area, this is primarily Medicare and Blue Cross Blue Shield. This project was created because of the stimulus of the Health Care Financing Administration's new prospective payment program for Medicare and has received continuing encouragement and support from the HCFA. Blue Cross and Blue Shield have also strongly supported the project."

Appended to Dr. Nelson's statement was a letter from Margaret Van Amringe, acting director of HCFA's Office of Survey and Certification.

"The HCFA assumes," she wrote, "that Medicare reimbursement for services provided in freestanding ambulatory surgery centers will result in a $2 million savings in fiscal year 1983 alone. . . . There is some evidence that the establishment of a freestanding surgical center in close proximity to a hospital has resulted in lower hospital fees for the same procedures. All in all, we are of the opinion that ASCs promote competition and thereby benefit both the Medicare program and its beneficiaries."

Then Dr. Nelson shot some holes in the State Medical Facilities Plan, on which Dr. Kenley had based his decision.

"We live in a fluid, flexible and innovative society," Dr. Nelson wrote, "and to hold in the year 1983 to a Plan written in 1979 is absurb and un-American. It was clearly the legislative intent that the Plan be updated appropriately, and this responsibility was clearly accepted by the Department of Health. The Department has been woefully neglectful. . .

"Physicians have been asked to do something about the high cost of medical care. We are trying. We are attempting to obtain a certificate of need for a facility that would significantly reduce the cost for many surgical and diagnostic procedures."

THE APPEAL OFFICER'S verdict was handed down one month later. Documenting his decision with six pages of pros and cons, Dr. Berrey found for the urologist's proposed surgery center. His reasons: 1) Cost savings "argue overwhelmingly in its favor." 2) The State Medical Facilities Plan's guidelines to determining the need for additional ORs "does not consider efficiency of operation." 3) The Plan "has not been updated to reflect recent changes in the reimbursement system, so that innovation and creativity are inhibited." 4) Blue Cross Blue Shield supports the proposed freestanding ASC because it has the potential for lower costs.

Altogether, Dr. Berrey concluded, the project may well bring greater efficiency to the system, foster competition and lower costs, and he asked the Commissioner of Health to set aside his denial.

In a covering letter, the Commissioner did just that, and he enclosed the object of Dr. Nelson's campaign—the official certificate of need for the urologists' freestanding ambulatory surgery center.

He remained concerned, Dr. Kenley noted, about the impact of the project on the existing operating room capacity in PD 15, but since third-party payers were encouraging the development of these ASCs, his approval of this pilot project might provide "an opportunity for the study of its effect on the health care system."

Dr. Nelson promptly began making his way through the sticky wickets of licensure for the Urology Center. He figured he'd get his hands on the necessary piece of paper without too much difficulty, and maybe the ASC could open for business in mid-December or thereabouts.

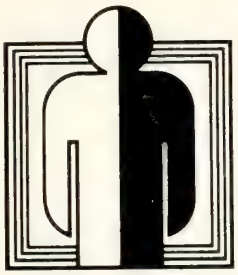
Up the street, construction finally ended on the new ambulatory surgery center at St. Mary's Hospital, and patients began to be scheduled in August. There are two dedicated operating rooms; St. Mary's had asked for four ORs, but the reviewers halved that number.

In Roanoke, the three operating rooms of the Lewis-Gale Clinic's ambulatory surgery center are being built into the third floor of a big new addition now under construction, with completion expected in the summer of 1984. The clinic is adjacent to Lewis-Gale Hospital, which was founded by physicians in 1908; today the clinic owns the emergency room and Hospital Corporation of America owns the hospital.

There's an outpatient surgery program at Lewis-Gale Hospital; the procedures are scheduled into a regular operating room. A spokesman was asked if the hospital intended to install a full-fledged ambulatory surgery center. "No, I don't think so," he said, "not now that the clinic is putting one in. I wish we'd gotten there first, because that's the way the business is going."

The author appreciates the generous assistance of the following persons: at St. Mary's Hospital, Christopher M. Carney; at the Lewis-Gale Clinic, Darrell D. Whitt; at Lewis-Gale Hospital, Robert D. Anderson; at the Central Virginia Health Systems Agency, Michael Osorio, Jack Quigley, Julie White and Anita Flint; at the Virginia Department of Health, Marilyn West, Elizabeth Taylor, Sue Rhodes and Steve Sorin of the Division of Resources Development, and May Fox and Deborah Blackburn of the Division of Health Planning.

THE HARD PART COMES AFTER THE DETOX



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From an alcoholic, a thank-you note

It is good to read Dr. Keiter's article on helping alcoholics, for he writes both knowingly and feelingly about alcoholism. It is good, too, that his observations on alcoholism appear here in Virginia Medical. We alcoholics hope fervently that all physicians would heed what Dr. Keiter says so eloquently.

I am a certified alcoholic. I know full well that few are the physicians who manifest patience with an alcoholic patient. Nor is mine an isolated feeling on that subject: I have listened to scores of fellow alcoholics during hours of discussions at hospitals, treatment centers, and Alcoholics Anonymous meetings.

My own experience with physicians is, I believe, typical. I was hospitalized half a dozen times for "nerves," for a faulty pyloric valve, for ulcers, for hypoglycemia, for depression. But no doctor ever confronted me with the fact of the underlying alcoholism until after I had moved out of my spirited denial and submitted myself to treatment.

That took years. Toward the end of those years I lost a 20-year marriage, a 25-year job. And I was arrested four times.

Of course, no physician ever poured a drink down me. But too many of them stood mute for too long, while I poured too many drinks down myself. They did not consult with family or employer; they did not recommend AA or institutional treatment.

It's not at all that I loathe physicians. I grew up in a mountain culture where doctors were dieties. Once instilled, dieties are hard to shake. And I'll admit that alcoholism is a hard-shelled diagnostic nut, and that I'm a stubborn cuss. But stubborn cusses need treatment, too.

"Despise the alcoholism, not the patient," says Dr. Keiter. Now there's a good saying. Too many doctors, many alcoholics feel, despise us. Or they are afraid of us because they don't know what to do with our problem.

Then there are the physicians who treat the drug of alcoholism with other drugs, the tranquillizers and the barbiturates. One doctor had me on five different drugs over a relatively brief span. My consumption of alcohol was reduced, but I was in a crazy daze.

Alcoholism now is classified as a disease. Perhaps it is, perhaps it isn't. Maybe the theory of genetic susceptibility is a myth. Maybe alcoholism comes from mental or spiritual weakness. Maybe alcoholism is an allergy, like an allergy to strawberries or tomatoes. But how many people with an allergy to strawberries hide a quart of them in the dirty clothes hamper and nibble away when nobody's looking? Or whoever got charged with driving under the influence of tomatoes?

Whatever it is, the "tough love" treatment prescribed by Dr. Keiter is respected by most of us alcoholics. And we are stimulated to higher hope in our observation that young physicians coming into practice seem to have a greater empathy for us. Maybe they won't be prescribing drugs to treat addiction to alcohol. Maybe their knowledge of alcoholism will extend beyond the ability to diagnose the cirrhosis and GI bleeding they come upon in their rounds.

We would wish it so. For there are tens of thousands of alcoholics out there. For some of them, early diagnosis, confrontation, and treatment can mean all the difference.

Thanks, Dr. Keiter, for writing about things like that. And thanks, Virginia Medical, for printing it.

–Carl L. Shires

Gary Brookins

Helping Alcoholics

Robert H. Keiter, MD, *Portsmouth, Virginia*

"Sometimes it is hard to remember, when treating these patients, that it could be ourselves under other circumstances and that the drinking caused the problems involved, rather than the problems causing the drinking."
—Francis J. Braceland, MD[1]

PHYSICIANS are so exposed to the dark side of alcoholism that it often seems incomprehensible that there are many alcoholics who actually recover, yet there are in every community groups of alcoholics in various stages of resolution of their addiction. Some of them fail completely, others reach a plateau of recovery which is at least tolerable, and some function marvelously well.

Out of my experience in treating alcoholics, I have formulated the concept of alcohol addiction as the equivalent of an acquired instinct. It is as though the alcoholic patient is driven instinctually to consume alcohol, recruiting intellectual resources and survival skills in the service of the addiction. I see this as an outgrowth of a pharmacological response to alcohol in which the alcoholic cannot control reliably the amount consumed or the behavioral outcome of the drinking. Loss of the capability to drink as one intends to drink is the elementary definition of alcoholism.[2] Susceptibility may be genetic.[3]

As physicians, we may attempt to counsel our alcoholic patients by appealing either to their intellects or their consciences. But the intellect subserves the addiction and is not allied to health issues, while the conscience is largely neutralized by the rationalizations supplied by the intellect. Any remaining guilt is completely dissolved with the next drink. We may insist to these patients that they are not functioning well, but alcohol gives them a convincing illusion of mastery and power.

Some patients become belligerent and protect their drinking rationale with a wall of hostility sufficient to deter the most enthusiastic of therapists. Others become masters of passive compliance, giving the appearance of cooperation and even appreciation, but shielding themselves from actual change.

There are those who use methods of "controlled" drinking to limit their intake. Staying away from "the hard stuff", counting drinks, using antianxiety drugs, repeatedly "going on the wagon" are some of these methods, which as a rule break down eventually, with further losses of time and health.[2]

Some also regularly seek detoxification so that they can drink comfortably again. Physicians and other clinicians often accommodate these strategies if no adequate therapeutic countermeasures are available. We become unwilling partners in the maintenance of the addiction process.[4]

From Maryview Psychiatric Hospital, 3636 High Street, Portsmouth VA 23707.

Submitted 1-21-83.

Even if the alcoholic patient becomes abstinent, he or she may refuse help for the emotional sequelae of alcoholism. Issues of resentment, guilt, and loss of intimacy with significant others are not addressed directly; instead, a prescription is sought as an anxiety remedy. The addict may seek somatic treatment for depression, but if guilt and personal isolation are not resolved, treatment is likely to fail. Efforts at individual psychotherapy may create a pathologically dependent relationship with the physician, within which the basic premises for drinking are carefully protected.[5]

THE American Medical Association now terms alcoholism a disease and offers guidelines for diagnosis, treatment and referral.[2] Alcoholics Anonymous has developed the essential concepts from which effective treatment programs have been formulated.[6] The disease terminology has been welcomed by many in the treatment field while offending others; it has also been referred to as only a disagreement in semantics.[7] But anyone with a "disease" is afforded special consideration in our society. Specific diseases require specific therapies.

The first treatment of this disease is pain. This must not be prescribed but is to be permitted within reasonable limits and must be attributed directly to the disease.

Emotional pain is most effective. Generally, emotional pain exists as a values conflict. A growing awareness in the alcoholic patient that his or her current behavior is in conflict with one or more

An impaired physician's story

I AM 50 years old and have not taken a drink for six years. This is a remarkable achievement for me, because I drank heavily for 30 years, losing in the process my family, my home, my medical career and my health.

I was born, reared and educated in Europe. My parents were separated when I was little, and my mother and I and a sister went to live with my maternal grandparents. I remember both my mother and my grandmother warning me strongly of the dangers of alcohol. I also remember the talk when a man in our town went to New York to be cured of drinking by some new method. Much later I learned this method was Alcoholics Anonymous.

In high school, many of the teenagers were experimenting with alcohol. I took my first drink at age 15 and liked its smell, taste and aftereffects. Drinking soon became a frequent diversion, and I had lots of fun without any problems.

In college, math and the basic sciences were my academic majors, and frequent drunkenness was my major extracurricular activity. I began having problems at school and, in the evenings, with the police, but I couldn't seem to do anything about it. I have saved a letter, written at this time to the young woman who was to become my wife but never given to her, in which I describe my frustration at not being able to stay away from drinking and stick to my studies.

I managed to get through college on schedule, but in the yearbook, in which each graduate was represented by a drawing and a poem, I was depicted wrapped around a lamppost with a bottle sticking out of my hip pocket, and the poem derided my drinking. That hurt, but it didn't stop my drinking.

I got married and entered medical school. There I was introduced to amphetamines, which enabled me to drink longer at night but still get up in the morning for classes. My main worry was in hiding my alcoholic breath; I used all kinds of mouthwashes.

In medical school we were taught about the bad effects of alcohol on the body. That worried me a lot, but it didn't deter my drinking.

To finish medical school, I quit drinking for a month or two before each exam and studied day and night. It was hellish, and after passing the tests I felt justified in going on a long drinking spree.

Entering general practice after internship, I got into all kinds of trouble, both at work and at home. Among other things, I wrecked two cars and lost my driver's license. Also, medical problems began to show up. After three years of this I decided to go to the United States. My wife and four children came with me.

At a private hospital in the Midwest I interned in internal medicine. I liked it there very much. At first I had difficulty keeping myself supplied with amphetamines, but then the drug companies began promoting diet pills and stuffed the mailboxes with samples. Most of the doctors had no

values, such as staying healthy, or keeping the family together, or retaining community status, will be accompanied by increasing "dis-ease".

It is physical or emotional pain that drives a person to seek help, and although alcohol is a temporary pain-killer, alcoholism has an inevitable progression towards a crisis of emotional pain—provided that those around the alcoholic desist from rescue efforts and allow this to happen. Each crisis is an opportunity for helpful intervention,[8] in which the physician has the particular role of confronting the patient with related health issues.

Clinicians who have resolved counter feelings toward alcoholic behavior will be the most effective interveners. Physicians who have had a close personal experience with alcoholism, such as with a parent, may have difficulty. Too much pain is bound up in these memories, and physicians with this kind of background need much help before they can experience a genuine concern untainted by anger or moral judgement.

Berating alcoholic patients is counter productive.[9] Informing them, in a climate of grave concern, that they are dying or at least facing calamitous loss of mental or physical health due to a disease beyond their control may get the attention of dormant survival instincts. This momentary concern may be quickly overridden by alcoholic logic. The alcoholic may say, "You've really scared me, Doc, I'll never touch another drop," i.e., "I'll take care of it." Firmly and with conviction the alcoholic must be told, "The disease of alcoholism is beyond

use for them, so I scooped up the discards. This met my needs until I went on the drug companies' mailing lists and could order for myself. To the amphetamine I added meprobamate and washed them down with alcohol. This was the main part of my diet for the next 12 years.

In internal medicine there was close contact with patients, which threatened to betray my drinking, so I decided to go into anesthesia, and I began a residency at a university hospital in another state. It was hard work, and my drug and alcohol abuse was continuous, so my physical and mental condition deteriorated rapidly. One day I was found unconscious on the hospital locker room floor. The Chief, a very kind man, had me taken to his office. When I woke up, he talked to me and offered me the chance to continue the residency if I would go into the hospital's psychiatric ward and have outpatient treatment after discharge.

I was happy to accept, but the hospitalization was a terrible time. Everything seemed to be falling apart. I felt so degraded and embarrassed and scared.

The experience kept me away from alcohol for a time, but the craving became too strong and I began to drink again, although I managed to complete my residency and get my medical license in three states. Then, back in practice again, this time in anesthesiology, my drinking became much worse. I was robbed and beaten, and once again lost my driver's license. Feeling completely broken down, I tried Alcoholics Anonymous. For a while that helped, but when I started to feel better, the meetings began to bore me—the same stories over and over again! Besides, I was a doctor and didn't need that stuff.

Back to drinking I went, so heavily that I could practice only a few days a week. One day I had a heart attack with cardiac arrest and suffered such damage that I have not been able to practice since. When I finally got out of the hospital and started drinking again, I became violent and realized I might harm someone in a drunken rage. I tried to shoot myself but didn't have the courage to pull the trigger.

In this journal, VIRGINIA MEDICAL, I saw an advertisement for an alcohol treatment place in another state. I took the ad to my doctor and asked him to check on it. When, after two months, he had done nothing, I called the administrator myself and made a date for admission. Arriving there, I was scared and nervous, but soon I came to like the treatment program, which was AA-oriented. Gradually things began to clear up. There were many lessons to learn, but it became easier to solve problems, some not favorably. After I had been sober for a year and a half, my wife and I separated, and that was the end of my family life.

On leaving the treatment center, I joined the local Alcoholics Anonymous and, a few months later, the International Doctors Alcoholics Anonymous, or IDAA, and I have been active in these groups ever since.

I am studying alcoholism and addiction and hope some day to find work in this field. Meantime, I do volunteer work for AA to try to help my fellow alcoholics.

H.V.G., MD

your control." Do not be dismayed that the addict does not believe this. The disease will do the convincing once the patient has struggled with it in vain.

It is important that the physician avoid expressions of frustration or anger. In the alcoholic, self-esteem rests on very shaky ground and there is marked sensitivity. If the physician is truly concerned about the alcoholic as a person, then the patient may respond as a person.

Despise the alcoholism, not the patient.

It is important to show these patients their laboratory reports, allowing them to see elevated enzyme levels, with explanations of the data's significance. Transient ketonuria, proteinuria, elevated uric acid levels, increased mean cell volume, increased gamma glutamyl transferase and other variations can be used as a basis for demonstrating the physical effects of drinking.[10] The metabolism of alcohol and its classification as a sedative drug can be explained.[11] Blood alcohol concentration, if previously obtained, may demonstrate tolerance and can be used in explaining the addiction process. Be sure to use a non-pejorative manner when communicating this information.

If the patient is not receptive, the physician might try to redefine the physician's role. For instance, I explain to these patients that I have a duty to inform them of my findings and will appreciate their attention so that I can discharge this obligation. This clarifies that impartial data is to be conveyed, not personal opinion.

With each office visit or hospital admission, the progression of the problem can be illustrated. Lack of success in controlling concurrent conditions, such as hypertension, diabetes mellitus, peptic ulcer, affective disorders, and so on, can be attributed to the alcohol. Frequently this involves the interaction of alcohol with prescribed drugs.[11] Patients can always escape to other physicians to avoid further confrontations, but the facts will haunt them. Many recovering alcoholics say that their drinking had been "ruined" for them long before they took decisive steps towards sobriety.

Once in a while the alcoholic patient will have suffered enough to be immediately receptive to the physician's message. The physician is then obligated to refer the patient at once to a program designed to treat alcoholism. Do not keep the patient waiting, lest alcoholic logic reassert itself. Ideally, the physician will have a facility in mind and will know if a bed is available. This will help if the patient is teetering with ambivalence.

Patient: "When will I have to go?"

Physician: "Today. I have reserved a bed for you."

Patient: "But I have to attend to important matters."

Physician: "Your health comes first."

Patient: "I have a lot of things that need my attention."

Physician: "You need to be completely sober so you can deal with them properly."

Patient: "Perhaps you're right."

This scenario may end differently, with the alcoholic insisting on putting off treatment. Be patient. The addiction will fulfill the physician's prediction, and the reality of it can be repeatedly illustrated—but with increasing concern rather than increasing anger.

Over time the alcoholic patient may become unable to believe old rationalizations, especially those for avoiding treatment, at which time a little encouragement and assistance with transportation may be all that is needed. Encouragement takes the form of hope in the knowledge that many people do recover, and the concept that an alcoholic is *what* the patient is, not *who* he or she is. *Never* destroy an alcoholic's rationalizations without delivering such hope and assistance. Otherwise you may create a situation which lends itself to suicide or other grave consequences.[11]

Remember to encourage treatment also for those alcoholics who have somehow remained abstinent for an extended period but are suffering intolerable anxiety and depression.[13] They need rehabilitation.

Likewise, those engaged in "controlled drinking" may accept treatment after it is pointed out that their drinking strategies are not necessary for non-alcoholics, nor are they conducive to restored health.

All these efforts may be stymied by the misplaced sympathy of other persons in the alcoholic's life. At meetings of Alcoholics Anonymous one hears that in the dictionary "sympathy" can be found between "shit" and "syphilis", a reference to the perception that sympathy can kill alcoholics.

Frequently, the alcoholic far advanced with dementia and other deteriorations is accompanied to the physician's office by a spouse who has nurtured the alcoholic along to this pitiable end. Alcoholics need "tough love" from those around them, so that they can suffer earlier the consequences of their drinking. Arrest, marital separation, financial crisis or whatever—these may be the painful events required to break the addict's denial. The physician's

role may involve assistance in pulling out the props, pointing out to all concerned the nature of the problem, then offering avenues of treatment and rehabilitation.

Occasionally one finds greater resistance and more intense anger in the family than in the alcoholic, and there can be such desperate determination by the family to control the situation that the physician is given no options. Recovery of the patient may threaten the stablity of a pathological family system wherein emotional pain binds the family into an inflexible structure.[12] The physician is challenged to be as patient and persistent with resistant family members as with the alcoholic.

Exposure to Alcoholics Anonymous, Al Anon and educational programs can be of immense help to the physician in understanding alcoholics and their interaction with their families. Some AA meetings are open to any person wanting to learn about alcoholism; these meetings, particularly those with a discussion format, furnish opportunities for insight into the alcoholic process. The book *Alcoholics Anonymous*,[14] is rich with autobiographical case histories and well worth reading.

Al Anon is a self-help fellowship independent of AA but with allied goals—to provide information, support and avenues of personal growth to relatives of alcoholics. Families and friends of the alcoholic patient should be encouraged to attend Al Anon meetings; it is helpful whether or not the alcoholic ever recovers. Referral of relatives to persons qualified in formal interventions is ideal. A well-rehearsed intervention by the family under such leadership is a powerful tool for bringing the alcoholic into treatment.

Perhaps nowhere in medicine is the quality of treatment as important as in the treatment of alcoholism. AA members can tell you about treatment facilities, although Alcoholics Anonymous itself makes no recommendations. Talk to someone with at least two years of sobriety.

For alcoholic patients who are financially destitute, Alcoholics Anonymous is an ever-present resource at virtually no cost. An AA representative will visit with any patient who has a desire to stop drinking and will help get the patient to meetings. Indigent persons may also receive counseling and support from state facilities and halfway houses.

Successes with these intervention and referral techniques are richly rewarding and reinforce new attitudes about this treatable condition. An improved working relationship between the medical community and alcoholism treatment programs will help control this major public health problem.

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Commentary: Emotional Problems of Doctors

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"We are expected to be strong, protective, wise, nurturant and available—a tall order indeed," observes the author, and one that can induce emotional disorder if the physician is not alert to the danger signs.

THE LITERATURE documents that doctors experience significant morbidity from substance abuse, depression and marital maladjustment. The exact extent of these problems is difficult to document because of under-reporting of physician mental illness and suicide. Studies estimate that over 4,000 physicians (1.5% of practicing doctors) are drug addicts.[1] Vaillant documented that 47% of physicians studied had bad marriages compared with 32% in a control population.[2] The best data suggests that the suicide rate for male physicians in America is the same as for age-matched controls, but that "female physicians have a suicide rate 3-4 times that of non-physician women . . . a rate similar to male physicians."[3]

It is uncomfortable for each physician to explore his or her degree of liability for mental illness. It is easier to deny that we have problems or to rationalize that our problems are no more severe than those of others and therefore are unimportant and will resolve with time or our own efforts. It is more productive to explore factors which stress physicians.

Even before the prospective doctor applies to medical school, both external (i.e., family) and internal motivators can cause anxious striving for grades to facilitate admission to a "good" medical school. Once admitted, the student often trades anxiety about getting in for anxiety about staying in. Studies have shown that many medical students receive treatment for emotional problems while in school.[4] Later in training, residency requires long hours and the process of dealing frequently with multiple services which require new skills and learning. The houseofficer has intimate contact with death and human suffering. Often the emotional and time demands of this training seriously damage marital relationships.

It is common at each level of training and practice to rationalize "if I only survive now, I will relax and take it easier later on." This unfortunately rarely happens because each subsequent stage has major challenges and stresses. The young physician in practice is often unwilling to turn away work, fearing that colleagues will find other referral sources. This can result in very long hours and reduced personal time. Many young physicians

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have little experience managing an office or financial affairs. Excessive financial commitments are easy to make when one has been poor for years, has little fiscal experience and has availability of credit. Some doctors find themselves working to pay for things they don't have the time to enjoy.

The public's expectation of doctors often originates in grossly unrealistic television portrayals of the doctor with the time to make tremendous personal investment in each case and find a way to save each patient from disaster. Our patients expect us to be strong, protective, wise, nurturant and available—a tall order indeed.

There are often increased stresses on female and minority medical students and physicians. They report subtle skepticism from colleagues and patients about their ability, which causes stress and which is often dealt with by time-consuming, energy-depleting overcompensation. Both groups continue to have few role models. All physicians must cope with conflicting pressures from home and work, but female physicians currently experience greater stress in this area.[3,5]

While these stresses are very important, they are only a part of physician morbidity. Most physicians handle the stress of medical practice by setting appropriate limits, and thereby thriving. Research and experience teaches that doctors are not a uniform sample of the general population.[6] We tend to have personality features which attract us to medicine and which may contribute to our success, but which may also facilitate maladjustment. These characteristics of the obsessive-compulsive personality include being hard-working, setting unrealistic and inflexible standards, having excessive anxiety and being very vulnerable to guilt. We tend to be "workaholics". George Vaillant studied students selected for good mental health over a period of many years. Those who became doctors were matched with a non-medical control group. The physicians who ultimately developed mental illness had characteristics of dependency, passivity, self-doubt, guilt and hypochondriasis.[2]

Some doctors are successful in early years only to encounter trouble later. The young physician may maintain a hectic schedule and his/her family may tolerate the regular denigration of family needs to those of the practice. But as time progresses, the physician may be less physically and emotionally able to maintain the original pace and indeed may have even greater responsibility, while the family may lose patience with continual low priority. Under these conditions, the physician's sense of work and life satisfaction may decrease, and vulnerability to depression, substance abuse and marital maladjustment may increase.

There are many protective measures available to physicians to reduce the likelihood of work-related emotional disorder. Maintaining flexible and reasonable goals is very important. As life changes the doctor must be able to change obligations and goals. Priority time for family and recreation sounds so easy, but in reality it takes great effort to maintain activities and relationships in these areas. Doctors must be aware of signs and symptoms of depression, which include insomnia, anorexia, weight loss, chronic pain, irritability and difficulty concentrating. Other danger signs include routinely making rounds at unusual times and developing a fear of having consultants review one's work.

Doctors should avoid treating themselves or members of their families. Unfortunately, we fear admitting problems and illnesses to ourselves or others. Many doctors feel it is weak and humiliating to consult a colleague for fatigue, depression, substance abuse or family problems. The physician is afraid of losing patients and fears being punished by the Board of Medicine. It appears easier to ignore the problem or to self-medicate. This unfortunate attitude results in poor diagnosis and inadequate treatment and allows the physician to decompensate to the point where serious damage to the practice becomes a reality.

Physicians and medical societies have become more invested recently in treating impaired physicians. The Medical Society of Virginia recently initiated just such a program. While it is often difficult initially to engage sick doctors in psychiatric treatment, the long-range results are usually gratifying. There is a great need for thorough and controlled current research of the emotional problems of physicians.

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Current Observations on Breast Abscess

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A study at the Medical College of Virginia reveals that the majority of breast abscesses treated over a five-year period occurred in non-lactating patients and that further breast infections were frequent in these patients. The authors discuss management of both the initial and recurrent infections.

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BREAST ABSCESSES are a relatively frequent occurrence. The classic presentation is described in a lactating female and the responsible organism is *Staphylococcus aureus*. The abscess is treated by simple incision and drainage and no further complications are expected.

Our recent experience, however, shows different patterns occurring in breast abscesses. The majority were noted in non-lactating patients and about 41% of the non-lactating patients with breast abscesses developed further breast infections. This prompted a review of our recent experience with this disease.

Materials and Methods

There were 35 female patients with breast abscesses treated at the Medical College of Virginia from 1975 to 1981. The age range of these patients was 19 to 62 years with a mean age of 35 years. All patients included in the study were initially treated by incision and drainage of the abscess. Patients found to have carcinoma associated with an abscess were excluded. Culture and sensitivities were obtained on 21 patients, and biopsies of the abscess wall were done in 23 patients. Slightly more than one-half of the patients received antibiotics in conjunction with the incision and drainage procedure. Five patients in the study were diabetic; all of these patients were not lactating at time of presentation.

Results

There were four patients who were lactating and two patients who were less than one year postpartum and in whom no specific mention of lactation was noted in their charts. These six patients comprised 17% of the total number of patients with breast abscesses. The mean age of the lactating patients was 23 years with a range of 19 to 27 years. All of the abscesses in this group were caused by *Staphylococcus aureus*. There were two patients in this group who required further breast surgery. One developed a mass at the incisional site seven months after incision and drainage of a breast abscess; an excisional biopsy revealed a chronic foreign body reaction secondary to a retained silk suture. The other patient developed in the incision a recurrent mass which was found to be a non-infected galactocele.

There were 29 patients who were not lactating at the time of initial presentation of the breast abscess. The mean age of these patients was 37 years with a range of 19 to 62 years. The abscesses occurred in the peripheral area of breast tissue in six patients

and the subareolar area in the remainder of the patients.

Of these 29 non-lactating patients, 12 patients, or 41%, developed recurrent infections manifesting as either a breast abscess or a draining sinus tract. (Since a number of these patients were lost to followup, this number may be even higher.) These recurrent infections were seen on the average of 10 months after the initial incision and drainage procedure, with a range of one to 36 months. Six (50%) of the patients who developed recurrent breast infections also had subsequent infectious complications of the breast requiring incision and drainage or ductal excision.

In these non-lactating patients the most common organisms isolated were *Staphylococcus epidermidis* (seven patients) and *Staphylococcus aureus* (seven patients). Other organisms sporadically cultured were *Proteus mirabilis, Enterobacteriaceae* species, *Streptococcus fecalis* and *Bacteroides fragilis*.

Discussion

Our data show breast abscesses requiring incision and drainage are much more frequent in non-lactating patients than in those who are lactating. This is similar to the observations made by Golinger in a review of mastitis and breast abscesses that 77% of his patients were not lactating at the time of presentation.[1] The organisms most commonly associated with breast abscesses in non-lactating patients are *Staphylococcus epidermidis* and *Staphylococcus aureus*, although numerous other pathogens were sporadically isolated in this study. In contrast, only *Staphylococcus aureus* was isolated in abscesses in lactating patients. Recurrent infections were relatively common, being seen in 41% of the non-lactating patients, but were not noted after incision and drainage in lactating patients. Two lactating patients did need further surgery for masses in the prior incisional site, however.

The high recurrence rate of breast infections in non-lactating patients after initial incision and drainage is consistent with recurrence rates noted by other authors.[2,3] The reason is most likely squamous metaplasia of the terminal mammary ducts, which causes obtruction from cellular debris and keratin.[3–6] This leads to dilatation of the duct and subsequent infection, which frequently presents in the subareolar area. Therefore, any treatment of the recurrent breast infection must include excision of the abnormal mammary ducts. Repeated breast infections may occur if excision of the obstructed mammary ducts is not performed. This is supported by the 50% incidence of subsequent breast infections in non-lactating patients seen in this study. Various authors recommend total ductal excision as the treatment of choice for recurrent breast abscesses due to the underlying mammary duct pathology.[1,3,7]

When confronted with a breast abscess, our recommendations are anti-staphyloccal antibiotic coverage with adequate incision and drainage of the abscess. At the time of surgery, a biopsy of the abscess wall should be done to rule out any associated carcinoma. The wound is then packed open to heal by secondary intention.

Close clinical followup is necessary in the non-lactating patient since recurrence is common. If recurrent abscess is noted, this is incised and drained. A total ductal exision as well as excision of the sinus tract is recommended once the inflammation has subsided. Frequently, a total ductal excision is done on the recurrent breast abscess at the time of the incision and drainage procedure. This allows definitive treatment of the ductal abnormality without a subsequent operation. Again, the wound is left open and packed with this approach.

The technique of total ductal excision is similar to that described by Urban, except that a circumareolar instead of a radial incision is used.[7] Once the incision is made, a subareolar flap is developed. The entire areola is then undermined and a cone of breast tissue including all the major ducts is dissected and removed. Only if there is no evidence of infection is the wound closed primarily; otherwise it is packed open.

We stress again that total ductal excision is necessary to achieve the best results with the least possibility of recurrent breast infections.

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Fence Post Impalement Injury of the Chest with Air Embolus: Case Report

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ALTHOUGH penetrating knife and missile wounds of the chest are relatively common,[1-4] impalement injuries are infrequently encountered and rarely reported.[5-9] The gruesome nature of impalement injuries can have a jarring emotional impact on emergency room staff. It is important, therefore, to take cognizance of the specifics so crucial to the management of this problem, as recently illustrated by the case of a patient brought into the University of Virginia Hospital emergency room impaled on a fence stake through the left chest after a single-vehicle automobile accident.

Case Report

A 34-year-old man was brought to the University of Virginia Hospital emergency room after his pick-up truck laden with diesel fuel containers skidded over an embankment during a heavy rainstorm. As his vehicle was coming down the embankment, it ran along and dislodged a fence; several fence posts pierced the windshield and penetrated the cab of the vehicle. Several stakes pierced the seat around the driver, and a single stake entered his left chest just beneath his clavicle in the midclavicular line (Fig. 1), exiting the thorax posteriorly in the sixth interspace three inches lateral to the vertebral column. The post elevated but did not break the skin posteriorly (Fig. 1 inset).

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Physical examination revealed a patient in moderate distress with stable vital signs and no evidence of other major injuries. In particular, there was no evidence of brachial plexus or major vascular injury.

Appropriate large-bore lines were inserted both for intravenous fluid infusion and for monitoring the filling pressures of the heart; tetanus toxoid and penicillin were administered, and the patient was taken to the operating room.

Under general anesthesia, a posterolateral thoracotomy was performed through the fifth interspace, and it was seen that the stake had impaled the upper lobe of the left lung but had not injured the major vessels of the pulmonary hilum or aorta. Under direct vision the stake was removed, and heavy venous bleeding was noted to be coming from the infraclavicular aspect of the entrance wound from the area of the subclavian vein.

A new heart murmur developed consistent with air embolus. The patient became hypotensive and was treated aggressively with ventilation, volume replacement, inotropic agents, and packing of the lacerated vein. The patient then stabilized, and after careful debridement and irrigation of the hemothorax, the lung laceration was repaired and the chest closed.

The patient's postoperative course was benign, and he was extubated the next morning and discharged from the hospital ten days later.

Discussion

Impalement injuries of the chest are unusual and require somewhat different management than that advocated for other penetrating thoracic injuries. Literature review revealed four similar cases, all of which were treated by thoracotomy, either to remove the penetrating object[5,9] or to repair the injured lung after the object was removed.[7,8] None of the patients in the literature had injury to the hilar or mediastinal vessels, and the impaling object was left in situ in three patients—both of which features undoubtedly contributed to the survival of these patients.

It should be noted that no matter where in the body an impalement injury occurs, it is critically important that the impaled object be left in the body and removed only in the operating room, where

there are facilities to take care of the resulting bleeding and to provide resuscitation if needed. Removal of the impaled object can lead to exsanguinating hemorrhage so profuse that death can occur quickly unless the proper equipment and personnel to deal with it are at hand.

A number of reports document the indication for thoracotomy after penetrating chest injuries.[1-4] Suspected or documented cardiac tamponade, continued blood loss, or an accelerated rate of blood loss through the chest tubes are usually accepted as indications for surgical exploration. Impalement injuries, although obviously in the "penetrating chest injury" category, should be managed separately, as they always demand urgent thoracotomy to remove the foreign body under direct vision, to explore the coelomic cavity thoroughly, to repair any injured parenchymal structures, and to debride the entrance and exit wounds adequately. Removal of the penetrating foreign body is often followed by major bleeding due to the loss of the tamponade effect. If more than one coelomic cavity has been violated, then a thoracoabdominal exploratory incision should be made. If the impaling object traverses both the right and the left pleural cavities, then a thoracotomy, perhaps of the left chest, should be made to assess and repair any mediastinal injuries and the right hemothorax could be drained by closed thoracostomy tubes.

Air embolism has not been previously reported with such impalement injuries. In lung injuries complicated by air embolism, the air usually enters lacerated pulmonary veins during artificial ventilation.[10] In this case, air entered the vascular system in the area of the subclavian vein in the infraclavicular region (the entrance wound). More expeditious packing of this area earlier in the surgical management of the patient might have prevented the air embolism. Systemic venous air embolism thus can be anticipated with penetrating thoracic injuries, as a potential cause of significant morbidity.[11]

Summary

Impalement injury of the chest is always dramatic and requires urgent thoracotomy, not only to allow removal of the foreign body under direct vision, but

Fig. 1. Patient intubated and ready for emergency thoracotomy with impaled fence post still in place. Inset: Arrow points to skin tenting up over point of impaled post at exit wound.

to facilitate aggressive debridement of the contaminated tissues and careful repair of all injured structures. Careful attention must be paid to all sites of blood loss, and prompt tamponade of any venous injuries is necessary to prevent air embolism.

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VIRGINIA AUTHORS

Traumatic Acute Subdural Hematoma: Major Mortality Reduction in Comatose Patients Treated within Four Hours. John M. Seelig, MD, Donald P. Becker, MD, J. Douglas Miller, MD, Richard P. Greenberg, MD, John D. Ward, MD, and Sung C. Choi, PhD, *Richmond.*

To discover which factors contributed to recovery after surgical intracranial decompression, the authors reviewed the records of 82 consecutive comatose patients with traumatic acute subdural hematoma (ASDH) who were treated in a single center under a uniform protocol. The delay from injury to operation was the factor of greatest therapeutic importance. Patients who underwent surgery within the first four hours had a 30% mortality rate, as compared with 90% in those who had surgery after four hours ($P<0.0001$). Other important prognostic variables included results of the initial neurologic examination, sex, multimodality-evoked potentials, and postoperative intracranial pressure (ICP). If all patients with traumatic ASDH were taken directly to hospitals equipped to diagnose and remove the hematoma within four hours of injury, mortality rates could be reduced considerably. N Engl J Med 304:1511–1518, 1981

Scrotal Palpation. David H. Zornow, MD, and Ralph R. Landes, MD, *Danville.*

Proper examination of the scrotum consists of inspection, palpation and, if indicated, transillumination. Inspection may reveal the hypoplastic appearance of an undescended testicle; enlargement caused by a testicular mass; the "bag of worms" signifying a varicocele; findings typical of torsion of the spermatic cord; or the "blue dot" sign, which indicates torsion of the appendix testis or appendix epididymis. During palpation, the testis must be differentiated from the epididymis. Am Fam Phy 23:150–154, 1981

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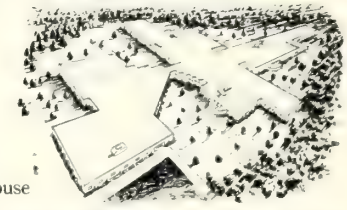

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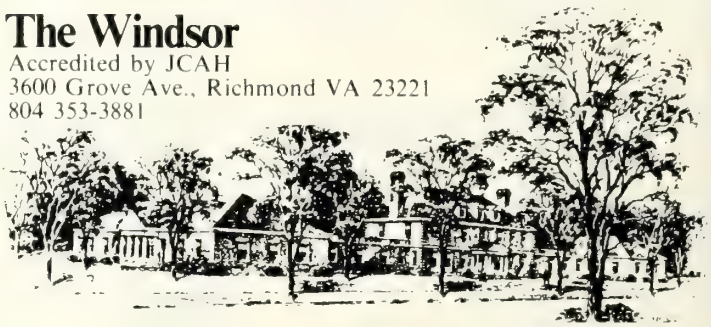

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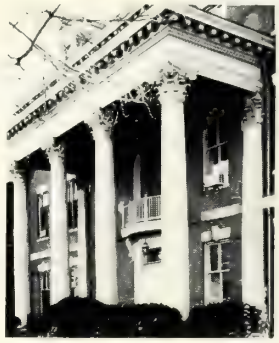

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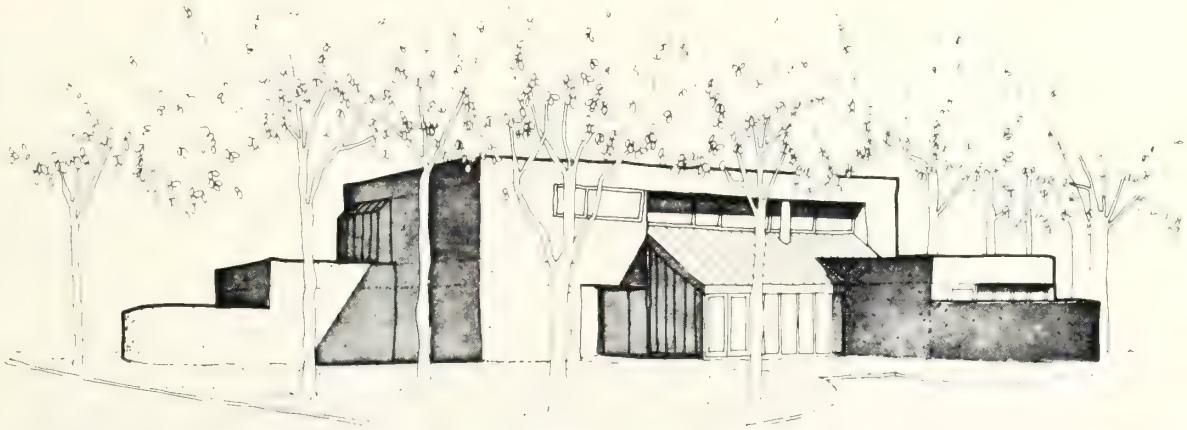

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VIRGINIA MEDICAL

EDITORIAL

"Survivors will be efficient, price-competitive and value-driven"

ELSEWHERE in this issue is a report of the approval of two certificate of public need applications for freestanding ambulatory surgical centers. While reports of CON "awards" and denials generally create no more than head-nodding in the medical and business community, these two latest approvals could have potentially far-reaching consequences. Now physicians, who have for the most part been entirely excluded from the process of having a financial say about costs, will be given new opportunities to become advocates of the patient with respect to quality and cost efficiency. More importantly, the shift in regulatory stance, in the face of excess capacity and increasing competition among providers, gives hospital and physician providers much needed incentives to price their services competitively while not limiting or denying the consumer the benefit gained through efficiency.

Blue Cross and Blue Shield of Virginia supported Dr. C. M. Kinloch Nelson's application and is convinced that more rational forms of insurance-benefit designs and a shift to prospective reimbursement will increase the demand for outpatient medical and surgical services. The relative value of these services is already apparent to the business payer and employee. How else can one explain the shift of support to more cost-effective and cost-conscious forms of health care coverage (e.g., HMOs and PPOs) and delivery (e.g., freestanding surgery centers)?

Heretofore, first-dollar coverage, regulation and cost-plus reimbursement placed undue emphasis on providing services and little if any emphasis on the *value*, or quality, of service. The business payer and the cost-sharing employee are no longer willing to accept rationalization of costs from providers, and we must accept the fact that value and service must go hand in hand. To appreciate the importance of the relationship of value and service as perceived by consumers-patients, I suggest reading *In Search of Excellence*, by Thomas J. Peters and Robert H. Waterman, Jr.

Changes in demands of the marketplace, regulation and reimbursement are putting strains on all parties connected with health care delivery. While the traditional relationship with Blue Cross and Blue Shield has changed for providers, it does not lessen the importance of the third-party payer and physician to objectively address the commensurate needs of the patient through equitable reimbursement agreements tied directly to sound medical practice. Fortunately, an opportunity exists for

both parties to regain the confidence of the patient-consumer in dealing effectively with health care cost. The future credibility of the medical profession and third-party payer rests on this single issue, a challenge which can be viewed either as a threat or an opportunity.

One fact is certain: Survivors will be efficient, price-competitive, and value-driven.

RICHARDSON GRINNAN, MD

Blue Cross Blue Shield of Virginia
PO Box 27401
Richmond VA 23279

"Let's turn medical costs back to competition"

THE 1970s found inflation out of control, and medical costs were included. Suddenly the most minor illnesses were costing astronomical sums. Ways were sought to slow the increase in the cost of medical care, and consumers and providers both looked for alternate methods of its delivery. They tried to decrease the duplication of services by establishing agencies to monitor cost and availability. Insurance companies encouraged less costly programs, and physicians themselves accepted and welcomed peer review. Cost-saving ideas, such as preadmission testing, one-day surgical stays and shortened length of hospitalization, were accepted by hospitals and physicians. In this atmosphere a federal law was passed requiring certificate of needs for certain expenditures. It was hoped that unnecessary increases in equipment, services and structures would be eliminated. Now, nine years later, it is time to reevaluate the CON program.

The certificate of need's main purpose is cost containment. During the past few years many of us who participated in the system have begun to question its effectiveness. Cost containment has not been realized and costs may have risen because of the system. The guidelines that a CON applicant must meet are usually out-dated. Since legal help generally is necessary to prepare and present a CON application, providers with cost-saving ideas may not pursue them. Those who do find it expensive and time consuming. The experience of Dr. C. M. Kinloch Nelson described elsewhere in this issue has happened to numerous other providers. Government agencies should be aiding those it serves and not discouraging new approaches to our problems.

The system is cumbersome, with too many levels. Despite this, the actual decision-making authority rests only with the Commissioner of Health, who is not bound to any of the health system agencies recommendations. If the CON system remains, it should be amended to give a committee of at least three people the final authority.

The newer methods of payment are the real reason that the certificate of need should cease. No longer will providers be reimbursed on a cost plus basis as newer payment plans, such as DRG's, are developed by the government and insurance companies. If a provider is able to offer a service at a lesser cost, then why should he have to seek a CON? He is not guaranteed a profit and competition will make him offer equal or less costly services. Free enterprise has realized this for years. The present system in fact encourages the establishment of monopolies in that it gives to a certain provider a lifetime license to offer a particular service.

The bureaucratic control of medical growth has been with us for some time and has failed. Let's turn medical costs back to competition and let those who can provide the best care for the least cost come forward without government hinderance. We will all benefit.

WYATT S. BEAZLEY III, MD

1805 Monument Avenue
Richmond VA 23220

"Frustrations of the past, hope of the future"

ALCOHOLISM is a perplexing problem for everyone concerned. Physicians, dedicated to caring for the sick and disciplined to confront the severest of human health needs, often find the treatment of the alcoholic a frustrating experience, which leads to the eventual conclusion that one is dealing with a totally hopeless situation and that such patients are not salvageable despite the most concentrated effort.

The non-professional looks upon alcoholics, if not actually as stigmatized persons, then at least as poor souls who are weak, degenerate, and without

the strength or courage to control their own destinies.

And the alcoholic patients are left without comfort or help, unable to cope with their disease or to find the solution needed to get them back into the mainstream of life.

But there may be a glimmer of hope.

The American Medical Association now defines alcoholism as a disease, subject to the same philosophical and therapeutic considerations as any other physiological disorder. Medical schools are initiating chemical abuse studies in their curriculua. And practicing physicians are becoming more involved to the extent that we now begin to hear the term "addictionologist" as a new and advancing specialty. Also, significant research is in progress suggesting a genetic link in at least some alcoholics.

There is encouragement also from the public sector, where AA and Al Anon and other educational programs have increased the intensity of their work, now including non-alcoholics in the battle. Industry has all but pointed the way to medicine through its very successful employee assistance programs.

And alcoholics themselves in some cases have begun to show that they can accept help when it is properly offered.

The ensemble of articles on alcoholism in this issue reflect all these frustrations of the past and the hope of the future. Dr. Keiter gently chides his colleagues for their indifference to the problem and then elegantly describes the methods that he has developed and the principle of "tough love" which he recommends to his fellow physicians. Carl Shires is even more critical of the profession, expressing his unhappiness, sometimes bordering on anger, at treatment that he has received—but then he recognizes the hope that Dr. Keiter is offering and applauds it. H.V.G. testifies that even after 30 years of illness, there is hope for recovery. Dr. Silverman illuminates the occupational hazards that may lead to a physician's impairment.

The members of The Medical Society of Virginia should look at these articles with more than curious academic interest. When the AMA became involved in the alcoholic program, it also asked us to look at ourselves, pointing out that physicians, just as others, have serious chemical abuse problems, and then declared emphatically that it is our ethical duty to seek out and help our colleagues who are afflicted by this disease.

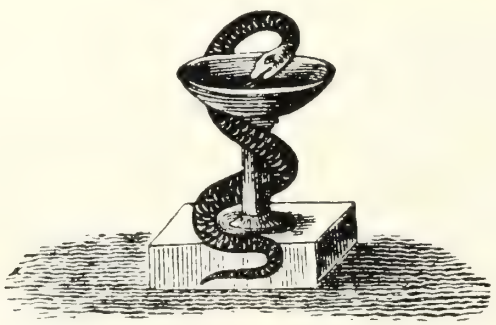
The Medical Society of Virginia, as is true of almost all state societies, followed that admonition with the establishment of its Physicians Health and Effectiveness Program, designed to identify, intervene, treat and rehabilitate our troubled colleagues. The program was adopted unanimously by the Society's delegates at Williamsburg last November and has attracted the help of many physicians throughout the state who have volunteered or been recruited to assist in this endeavor. Manpower is an important ingredient in the success of the impaired physicians program in Virginia and many more than those who have volunteered are now needed. Hopefully, physicians who have an interest in this field will provide their services.

In the beginning the Physicians' Health and Effectiveness Program had two goals. First, to improve patient care and second, to preserve the medical careers of our colleagues. Both objects are to be accomplished by one procedure—the removal from practice and getting impaired physicians into a treatment facility before they become threats to their patients or to themselves.

In view of the articles published in this issue, there may be a third dividend: As we seek to help ourselves, we may develop a better understanding, a deeper compassion, and a more dedicated therapeutic approach to our similarly impaired patients.

WILLIAM H. BARNEY, MD
1935 Thomson Drive
Lynchburg VA 24501

Dr. Barney is chairman of the Physicians' Health and Effectiveness Committee.



VIRGINIA MEDICAL OBITUARY

W. Frederic Delp, MD

Dr. W. Frederic Delp, longtime Pulaski physician and past president of the Southwestern Virginia Medical Society, died September 10 at age 77. He retired a few years ago because of ill health.

Dr. Delp was a native of Rural Retreat, Virginia, and he lived there all his life, leaving only for his education and medical training. He was graduated from Washington and Lee University and the Medical College of Virginia, Class of 1934. He received training at the University of Michigan, Ann Arbor, and returned to his hometown to begin a 26-year family practice in nearby Pulaski. Prior to his retirement, he served for seven years on the staff of the Veterans Administration Hospital in Salem.

A member of the Southwestern Virginia Medical Society, Roanoke Academy of Medicine and American Academy of Family Physicians, Dr. Delp had been a member of The Medical Society of Virginia for over 44 years.

Joseph W. Stein, MD

Dr. Joseph W. Stein, retired Arlington otolaryngologist, died November 9, 1982, at the age of 74. A native of Nashville, Tennessee, Dr. Stein was graduated from the University of Tennessee; trained in Los Angeles, Memphis, and Washington, DC; and served with the US Army during World War II. He had been a member of The Medical Society of Virginia and the Arlington, Fairfax and District of Columbia Medical Societies.

Memoir of James T. Tucker 1896–1982

By Paul D. Camp, MD, John P. Lynch, MD, and Charles M. Caravati, MD

Dr. James Thomas Tucker, a distinguished orthopedic surgeon, died in Richmond on July 14, 1982 at the age of 86.

A native of Richmond, he was graduated from the University of Richmond in 1923 and received his medical degree from the Medical College of Virginia in 1927. After a year at Walter Reed Hospital in Washington DC, he was a post-doctoral medical student at Harvard for two years. While in Boston he was at Children's Hospital and Massachusetts General Hospital. He did additional work in Austria.

He returned to Richmond and became associated with Drs. William Tate Graham and Donald Faulkner in the practice of orthopedic surgery. Later he was the senior member of Virginia Orthopaedic Associates and practiced with them until his retirement in 1977.

Dr. Tucker was emeritus associate professor of orthopedic surgery at the Medical College of Virginia. For years he was on the surgical staff of Crippled Children's Hospital, now Children's Hospital, and was surgeon-in-chief from 1955 to 1970. He was also a member of the Board of Trustees of Children's Hospital and a consultant surgeon for the Seaboard and Norfolk and Western railroads.

Dr. Tucker was a member of the Richmond Academy of Medicine, the Medical Society of Virginia, the Southern and American Medical Associations, American College of Surgeons and Academy of Orthopaedic Surgeons. A past president of the Virginia Orthopaedic Society, he was a diplomate of the American Board of Orthopaedic Surgery, fellow of the American College of Surgeons, fellow of the International College of Surgeons, and fellow of Societé Internationale de Orthopaedique et de Traumatologie.

A veteran of both world wars, Dr. Tucker was a retired lieutenant commander in the US Navy Medical Corps. He was active in the MCV Alumni Association and served on the board of trustees of the University of Richmond from 1947 until his death. The university conferred an honorary degree on him in 1973. An ardent horseman, Dr. Tucker rode regularly with the Deep Run Hunt.

He is survived by his wife, Katherine Huff Tucker.

Prepared and published at the request of the Richmond Academy of Medicine.

WHO'S WHO

Ask **Dr. Kinloch Nelson** of Richmond how his golf game's going these days and he gives you a fearful hard luck story. "I'm getting worse every year," he grumbles, "and I don't want to talk about my score."

Bosh. The 80-year-old medical education administrator shot his sixth hole-in-one on the 161-yard, 14th hole of the Country Club of Virginia's James River course on July 7, 1983.

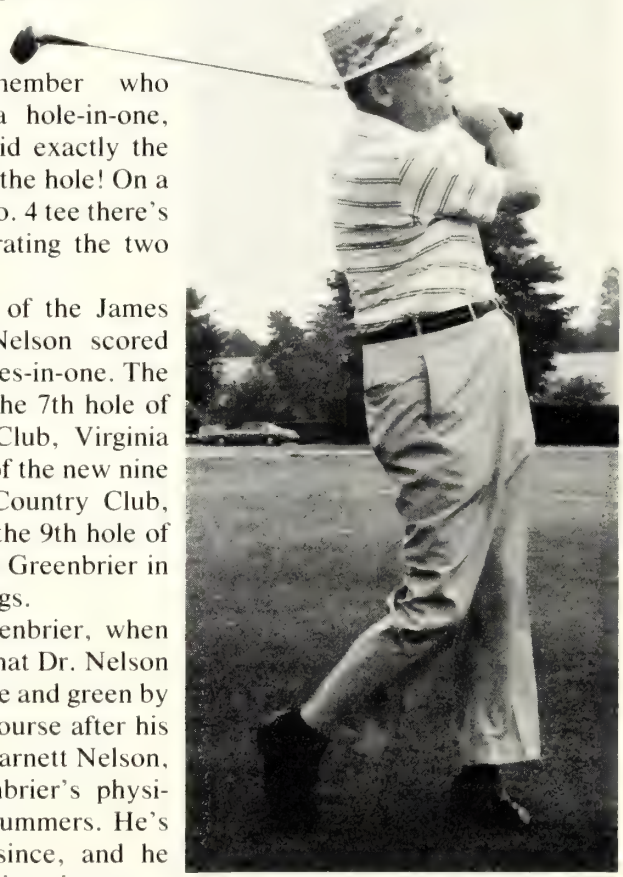
About 30 years ago, on that same course but a different hole (No. 4), Dr. Nelson and Richmonder Landon Cabell pulled an amazing stunt while playing a competitive round against each other. One of them (Dr. Nelson can't remember who played first) fired a hole-in-one, and the other one did exactly the same thing, to halve the hole! On a stone bench at the No. 4 tee there's a plaque commemorating the two aces.

On the 17th hole of the James River course Dr. Nelson scored another of his six holes-in-one. The others occurred on the 7th hole of the Princess Anne Club, Virginia Beach; the 8th hole of the new nine at the Farmington Country Club, Charlottesville; and the 9th hole of the old course at the Greenbrier in White Sulphur Springs.

It was at the Greenbrier, when he was 7 years old, that Dr. Nelson learned the joys of tee and green by tagging around the course after his father, the late Dr. Garnett Nelson, who was the Greenbrier's physician for a series of summers. He's been playing ever since, and he doesn't consider hitting six aces a big deal. After all, he'll tell you, when you've been playing golf for 73 years you're supposed to know where the ball is going.

Long identified with quality medicine in Virginia, Dr. Nelson is former dean of the medical school at the Medical College of Virginia and now directs The Medical Society of Virginia's continuing education program. A nephew, Dr. C. M. Kinloch Nelson, is the star of the cover story in this issue of VIRGINIA MEDICAL.

Five Medical Society of Virginia members have been selected by Gov. Charles S. Robb to serve in state posts.



Holing out: Dr. Nelson
Cyane Lowden photo

Appointed to the State Board of Medicine were **Dr. James B. Blayton,** Williamsburg; **Dr. G. Edward Calvert,** Lynchburg; **Dr. Barbara A. Mella,** Fairfax; and **Dr. Charles N. VanHorn,** Norfolk.

Dr. William M. T. Forrester, Richmond, was named a member of the Virginia Highway Transportation Commission.

In Kilmarnock, **Dr. Robert E. duPrey** is looking at the world through rose-colored glasses now that **Dr. Todd W. Geisert** has joined him in the practice of ophthalmology. Dr. Geisert recently finished his residency at the Medical College of Virginia.

Dr. L. Garcia, Williamsburg, has been appointed a fellow of the American Psychiatric Association. Dr. Garcia is director of psychiatric residency training at Eastern State Hospital.

New president of the Norfolk Academy of Medicine is **Dr. Lawrence A. Bernert, Jr.** Serving with him are **Dr. George H. M. Rector,** vice president, and **Dr. Jack D. Mahan,** secretary.

Dr. Edward E. Gahres, Alexandria, was elected president of the Virginia Obstetrical and Gynecological Society at its spring meeting, and these were elected to serve with him: **Dr. Clifford H. Fox,** Charlottesville, president elect; **Dr. John A. Board,** Richmond, vice president; and **Dr. Thomas A. Wash,** Newport News, secretary-treasurer.

Dr. J. Shelton Horsley III, Richmond, is the new president of the American College of Surgeons, Virginia Chapter.

The Virginia Chapter, American College of Emergency Physicians,

has named **Dr. Stanley E. Heatwole,** Staunton, as its president.

New president of the Virginia Society of Anesthesiologists is **Dr. Waverly M. Cole,** Richmond.

Dr. Stuart Ashman, Virginia Beach, has been elected to the board of directors of the Federation of American Hospitals. Dr. Ashman is medical director of Tidewater Psychiatric Institute in Norfolk and Virginia Beach.

With a goal of $250,000, staff physicians of the Stonewall Jackson Hospital in Lexington set out this spring to raise funds for the hospital and wound up way over the top, at $292,020.47. **Dr. Robert P. Irons** was chairman of the campaign, with **Dr. W. Barton Dick** and **Dr. O. Hunter McClung, Jr.,** helping him as vice chairmen.

Off the presses is the Fourth Edition of the classic *Disorders of the Respiratory Tract in Children,* edited by **Dr. Edwin L. Kendig, Jr.,** Richmond, and **Dr. Victor Chernick,** Winnipeg, Canada. The publisher is the W. B. Saunders Company, Philadelphia.

These Medical Society of Virginia members contributed to the new volume: **Dr. James W. Brooks, Dr. Barry V. Kirkpatrick, Dr. F. Stanford Massie, Dr. Dawn G. Mueller** and **Dr. Arnold M. Salzberg**. All are on the faculty of the Medical College of Virginia, where Dr. Kendig himself is a professor of pediatrics. He is also Editor of VIRGINIA MEDICAL. Dr. Chernick is professor of pediatrics at the University of Manitoba.

On his retirement at the end of the University of Virginia's academic year, **Dr. William N. Thornton, Jr.,** was elected professor emeritus. A native of Courtland, Virginia, Dr. Thornton received both BS and MD degrees from the University of Virginia and was chairman of its Department of Obstetrics and Gynecology from 1950 to 1979.

In Norfolk, **Dr. William P. Edmondson,** internist, was elected president of the medical staff of Medical Center Hospitals, and in Alexandria, **Dr. Michael Friedman,** radiologist, became the new president elect of the Alexandria Hospital's medical staff.

His recent retirement as director of pathology at Winchester Memorial Hospital set **Dr. George H. Murphy** to reminiscing on how much the hospital has grown in the 35 years of his service there. For instance, he told a newspaper reporter, when he joined the staff, "there were just three technologists with me. Now the hospital has 60 employees in the lab and four pathologists."

A Canadian by birth and an American by adoption, Dr. Murphy took his medical degree at Dalhousie University in Halifax, Nova Scotia, then spent five years during World War II in the Canadian Army in Europe, where he commanded a mobile bacteriology lab. After the war he trained at the Mayo Clinic.

He will not be completely retired, he told his interviewer, since he intends to be a consultant in laboratory management and plans "to play golf and spend more time with my ham radio and photography equipment."

The national Society of Teachers of Family Medicine this year gave its highest award, the Certificate of Excellence, to three of its members, and two of them were Virginians: **Dr. B. Lewis Barnett** of the University of Virginia School of Medicine and **Dr. Fitzhugh Mayo** of the Medical College of Virginia. Both are chairmen of their departments, and both have been recipients of the similarly prestigious Thomas W. Johnson Award of the American Academy of Family Practice, Dr. Mayo in 1974 and Dr. Barnett in 1976.

Dr. Richard E. N. Sedwick, Harrisonburg, was inducted as a fellow of the American College of Obstetricians and Gynecologists during its annual meeting in Atlanta.

For the second consecutive year **Dr. Peter Armstrong, Jr.,** Charlottesville, was selected for the Robley Dunglison Teaching Award by the graduating class of the University of Virginia School of Medicine. A 1963 graduate of London University's Middlesex Hospital Medical School, Dr. Armstrong joined the UVa faculty in 1977 and is professor of radiology.

"The **Howard and Georgeanna Jones** Institute for Reproductive Medicine." That's the new name of Eastern Virginia Medical School's Division of Reproductive Endocrinology, and it honors the two EVMS professors who have co-directed the pioneering in vitro fertilization program at EVMS. The Institute will be a subdivision of the Department of Ob-Gyn.

To **Dr. Frank B. Clare,** Portsmouth neurosurgeon, went the title of 1982 Sportsman of the Year given by the Portsmouth Sports Club. The honor signified Dr. Clare's 20 years of treating injured high school athletes in the Portsmouth area and his long tenure as team physician for the Churchland High football team. He has also helped conduct clinics for high school coaches. A retired captain in the US Navy Medical Corps, Dr. Clare was himself a quarterback for his high school team in Aurora, Illinois, where he also ran track.

VIRGINIA MEDICAL CLASSIFIED

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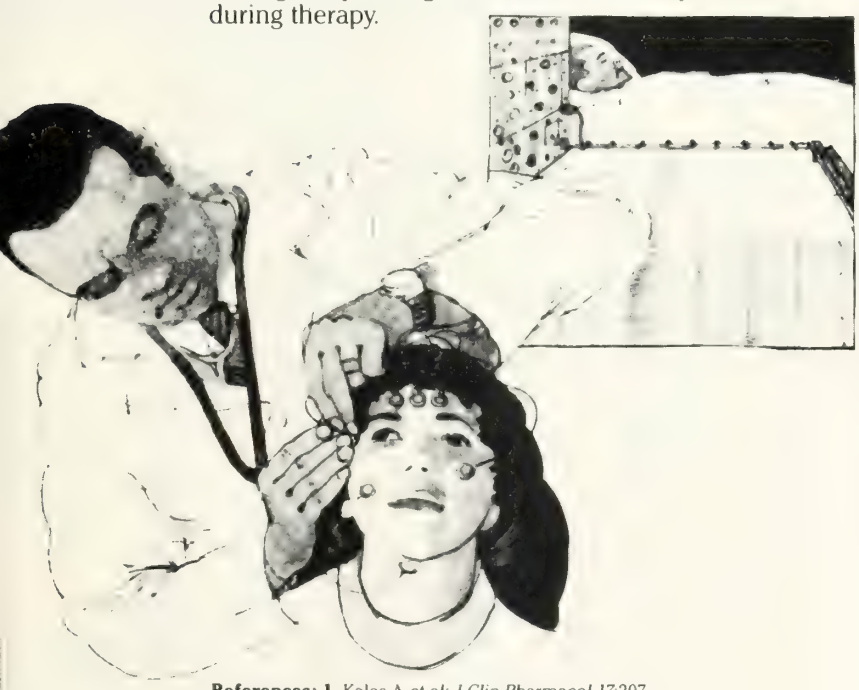

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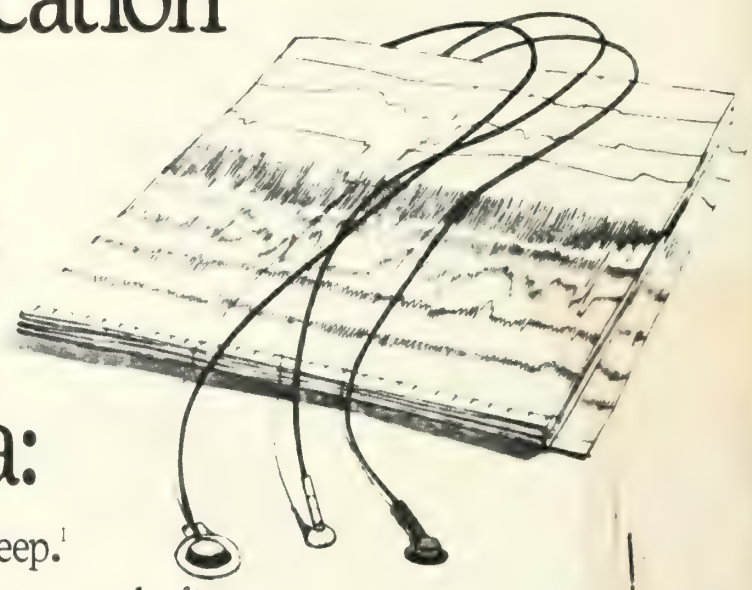
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The Award Winning Publication of The Medical Society of Virginia

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Old folks' needs rejuvenate a tradition

HOUSE CALLS seem to be making a comeback. Partly it's a matter of competition, says one Virginia physician whose house calls are on the increase—all those home health care agencies setting up shop, for instance. Partly it's the new third-party reimbursement, which encourages patients to ask for house calls. And partly it's because in Virginia, as all over America, the aging population is on the increase, and it's the elderly patient who precipitates the great majority of house calls.

Whatever the reasons, the requests for house calls are markedly on the rise in many Virginia doctors' offices.

"We just can't take care of them all," said one young woman who schedules the appointments of a Richmond GP group. "We take care of our regular patients, but we've had to become awfully selective about new patients who ask for house calls. I spend a lot of time trying to find ways to get patients into the office."

An internist's secretary reported fielding more requests for house calls recently—"and," she noted, "they don't want anyone but a doctor."

Cyane Lowden

Dr. Merrill heads for a house call.

Into this situation steps Dr. Cynthia W. Merrill of Richmond with her Physician home Visits, Inc., an enterprise that may be unique in Virginia.

"It was in early spring this year when I received a call from a lady who asked if I would come to see her elderly mother," Dr. Merrill recalls. "She said she had called 15 doctors before she called me. I went. That started me thinking, and I set up business a few months later."

Putting her money where her convictions were, she invested $6,000 in medical equipment, legal fees and advertising, and a small but steady stream of calls started coming in.

Her fee is $64 per visit, plus mileage for trips exceeding ten miles. "I don't think that's out of line," she says, "considering the time involved and also that an ambulance service would charge $60 each way for a trip to an office or hospital." Of the fee, Medicare will pay $36, and a major medical insurance policy will reimburse at the rate of 80%.

Most of the patients she has seen are elderly; in fact, she has visited only one person under 65. Most are women, bedridden and living with family or an attendant. In her bag she packs the standard examining equipment, including Hemoccult® and catheter sets. Her EKG machine represents $1,900 of

VSB BUREAU

THE FRANCIS

the cost of going into the home-visit business. If the need ever arises, she can call for a portable X-ray machine, but she hasn't needed it yet.

About one-third of the patients she has seen have had no doctors to call their own. In regard to the others, Dr. Merrill is sensitive to professional ethics and works carefully to preserve patient allegiance to the primary doctor. Callers are queried carefully for possible conflicts of medical interest, and on occasion Dr. Merrill has turned down a request for a visit when she felt it might be interpreted as an intrusion into another doctor's territory.

Thanks to this vigilance, she has heard nothing negative from her peers, she relates with relief, and some of them have offered support for her effort. "A couple of retired doctors called to say they would help out if I needed them. Others have told me they would refer patients to me for home visits, and I've had calls of thanks for the patients I've referred."

Asked for his opinion, a Richmond GP who makes house calls himself gives Dr. Merrill's service high marks. "It's a necessary service and a very good thing," he observed, "and it will improve the profession's image."

Like other physicians, Dr. Merrill screens calls to her service for emergency situations, and she is on the alert for drug addicts. She takes with her no drugs, only a prescription pad, and no needles, only vacutainers.

Physician home Visits, Inc., has given its proprietor an understanding of why house calls fell out of medical practice. "The visits are time-consuming," she admits, "and sometimes I find myself doing such things as helping a patient to the bathroom." And the home setting leaves a lot to be desired clinically. "Examinations are difficult, and of course I can't do a pelvic without stirrups."

On the other hand, she is quick to point out, the home setting promotes a closer relationship with the patient, and she finds the patients much more communicative there. Some of them, indeed, are quite desperate to communicate, she observes.

A native of New Jersey, Dr. Merrill was graduated from Ohio's Antioch College, where she majored in biology, and from the University of Virginia School of Medicine, Class of 1973. She trained at her alma mater and in Baltimore, and practiced in Hopewell and Highland Springs before opening her office on Richmond's Monument Avenue. Her husband, Dennis, is an assistant attorney general for the State of Virginia, and they have two sons, aged 5 and 9.

As backup for her schedule, Dr. Merrill has engaged on a part-time basis another family practitioner, Dr. Susan Schufeldt, herself a wife and mother.

THERE'S A Virginia physician whose practice consists entirely of house calls. His name is Dr. Griffith B. Daniel, and he is the director and sole attending physician of the City of Richmond's Home Care Unit, a municipal medical service thought to be unique in Virginia.

DR. HOLLAND TO SENATE

Dr. Clarence A. "Clancy" Holland, Virginia Beach GP and Medical Society of Virginia member, won a stiff, three-way contest in the November elections and will take a seat in Virginia's Senate when it convenes in January. He is a Democrat, as is his brother, Sen. Richard J. Holland, Isle of Wight.

The goal of the unit is to take care of the medical needs of every Richmond citizen who can't pay for the care. Dr. Daniel must visit and evaluate every new patient, an ambitious schedule, especially since new-patient requests are "ballooning," as one staff member put it. The case load of those receiving continuing care through the unit hovers around 300 persons.

Dr. Daniel is helped by two public health nurses, a nurse practitioner and a nursing aide, and he has on call several physicians in private practice to whom he can refer patients for continuing care when necessary.

The unit's patients are primarily the elderly, but there are some youngsters with handicaps. It's a preventive program: Without intervention at home, many of these patients would have to be hospitalized or even institutionalized.

A native of Louisa County, Dr. Daniel conducted a general practice there for 20 years before taking the municipal post. He is a graduate of VPI and the Medical College of Virginia, Class of 1950.

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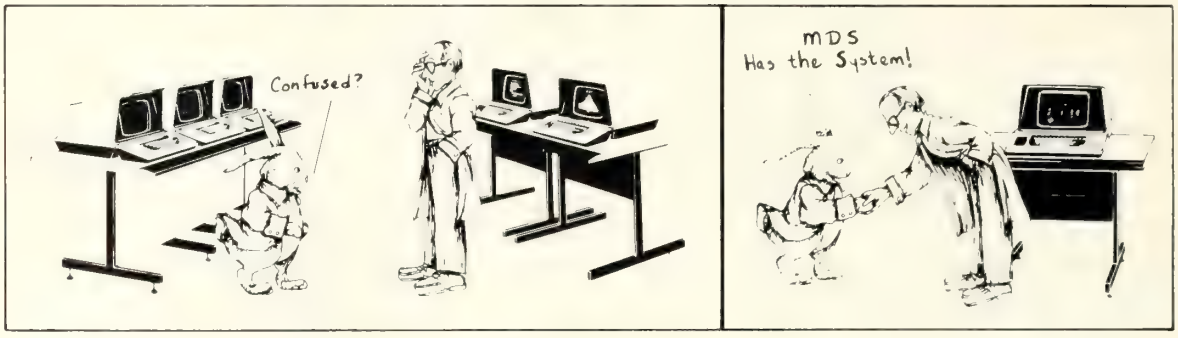

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LETTERS

A hearty commendation for the club that came to the rescue

Attempts over the past 20 years to get my hospital, community and medical society to join in an effort to honor Dr. Jessee Bennett were fruitless. As described in Dr. Wyndham Blanton's *Medicine in Virginia* and a previous article by me in VIRGINIA MEDICAL,[1] this physician did the first successful caesarean section in the new world.

Resolutions to The Medical Society of Virginia were approved and forwarded to the State Highway Department but were not successful in providing a highway marker to commemorate the heroism of this physician and his wife, the patient.

A plea to the local hospital, Rockingham Memorial, to name the new obstetrical unit, recently completed, in honor of these people was ignored.

In Kentucky a medical hero, Dr. Ephraim McDowell, has been honored and widely respected. His medical "first" was accomplished many years after that of Dr. Bennett.

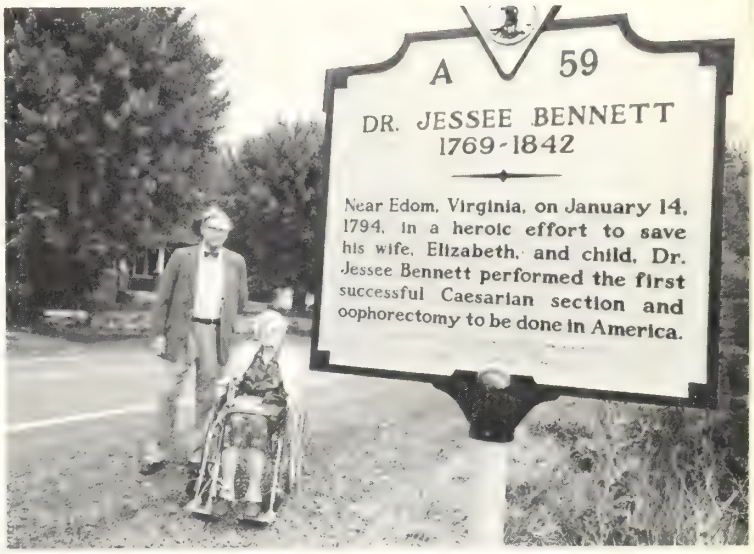
However, women to the rescue! The Linville-Edom Extension Homemakers Club determined that the story of this deed should be preserved for the future. Through great effort these ladies raised a considerable sum of money and paid for a suitable highway marker that has been erected in Edom, Virginia, near the site of the Bennett's cabin.

What organized medicine did not do these women did well. I wish to commend them with all my heart for their dedication in memorializing this great event in Virginia medicine.

George M. Nipe, MD

1031 South Main Street
Harrisonburg VA 22801

1. Nipe GM: "Jessee Bennett decided to operate on his wife at once." Va Med 106:885-885, 1979


A 59
DR. JESSEE BENNETT
1769-1842
Near Edom, Virginia, on January 14, 1794, in a heroic effort to save his wife, Elizabeth, and child, Dr. Jessee Bennett performed the first successful Caesarian section and oophorectomy to be done in America.

Beside the marker: Dr. Nipe and Mrs. J. O. Beard, Homemakers Club historian

Urges reconstruction for mastectomy patients

Why oppose post-mastectomy reconstruction?

Anyone who treats patients with breast cancer realizes that radical mastectomy has been supplanted by modified radical in most cases. It in turn is now being challenged by some who feel that sometimes a partial mastectomy with axillary dissection and radiation is adequate for local control. While we await a consensus on how best to achieve local control of this disease, most surgeons perform the modified radical mastectomy. One of the advantages of this operation is that excellent post-mastectomy reconstructive results can be obtained. This has been described in VIRGINIA MEDICAL and other journals.

As a general surgeon, I am surprised that, despite this, a number of post-mastectomy patients have been forbidden to undergo reconstruction because their physicians or surgeons feel it would make the detection of local recurrence more difficult. In fact it has never been shown that post-mastectomy reconstruction impedes or delays detection of local failure. In light of that, I would urge that the physicians of Virginia allow reconstruction for the one-quarter to one-third of mastectomy patients who desire it.

George A. Knaysi, MD

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CONTRAINDICATIONS: Hypersensitivity to any of the components, concurrent MAO inhibitor therapy, severe hypertension, bronchial asthma, coronary artery disease, stenosing peptic ulcer, pyloroduodenal or bladder neck obstruction. Do not use in children under 12 years.

Do not use this drug in patients with narrow-angle glaucoma, obstructive or paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrage, severe ulcerative colitis, toxic megacolon complicating ulcerative colitis, myasthenia gravis. Do not use in nursing mothers.

Use in treating lower respiratory tract symptoms, including asthma, is contraindicated.

WARNINGS: Caution patients about activities requiring alertness (e.g., operating vehicles or machinery). Antihistamines are more likely to cause dizziness, sedation, and hypotension in elderly patients. Patients should also be warned about the possible additive effects of alcohol and other CNS depressants.

Usage in pregnancy: Safe use in pregnancy has not been established. Use only when the potential benefits have been weighed against the possible hazards to the mother and child. Note that an inhibitory effect on lactation may occur.

PRECAUTIONS: Use with caution in patients with a history of bronchial asthma, increased intraocular pressure, hyperthyroidism, cardiovascular disease, hypertension, hiatal hernia with reflux esophagitis, intestinal atony of the elderly or debilitated intestinal obstruction, myasthenia gravis, renal function impairment, and ulcerative colitis (severe).

Drug Interactions: MAO inhibitors, Alcohol or CNS depressants, especially anesthetics, barbiturates, and narcotics.

ADVERSE REACTIONS: Prolongs the response to nervous stimulation, potentiates the response to norepinephrine, and inhibits the response to tyramine.

Slight to moderate drowsiness occurs relatively infrequently with Chlorpheniramine Maleate. Other possible side effects common in antihistamines in general include perspiration, chills, dryness of mouth, nose and throat.

Cardiovascular System: Hypotension, headache, palpitations, tachycardia, extrasystoles.

Hematologic System: Hemolytic anemia, thrombocytopenia, agranulocytosis.

Nervous System: Sedation, dizziness, disturbed coordination, fatigue, confusion, restlessness, excitation, nervousness, tremor, irritability, insomnia, euphoria, paresthesias, blurred vision, diplopia, vertigo, tinnitus, acute labyrinthitis histeria, neuritis, convulsions.

Gastrointestinal System: Epigastric distress, anorexia, nausea, vomiting, diarrhea, constipation.

Genitourinary System: Urinary frequency, difficult urination, urinary retention, early menses.

Respiratory System: Thickening of bronchial secretions, tightness of chest and wheezing, nasal stuffiness.

DOSAGE AND ADMINISTRATION: Dosage should be individualized according to the needs and response of the patient. Adults: one capsule every 8 to 12 hours not to exceed 3 capsules daily. Not for use in children under 12 years of age.

OVERDOSAGE: Treatment of the signs and symptoms of overdosage is symptomatic and supportive. In the event of overdosage, emergency treatment should be started immediately.

Treatment: The patient should be induced to vomit, even if emesis has occured spontaneously. Vomiting by the administration of ipecac syrup is a preferred method. However, vomiting should not be induced in patients with impaired consciousness. Stimulants (analeptic agents) should not be used. Vasopressors may be used to treat hypotension. Short-acting barbiturates, diazepam or paraldehyde may be administered to control seizures. Hyperpyrexia, especially in children, may require treatment with tepid water sponge baths or a hypothermic blanket. Apnea is treated with ventilatory support.

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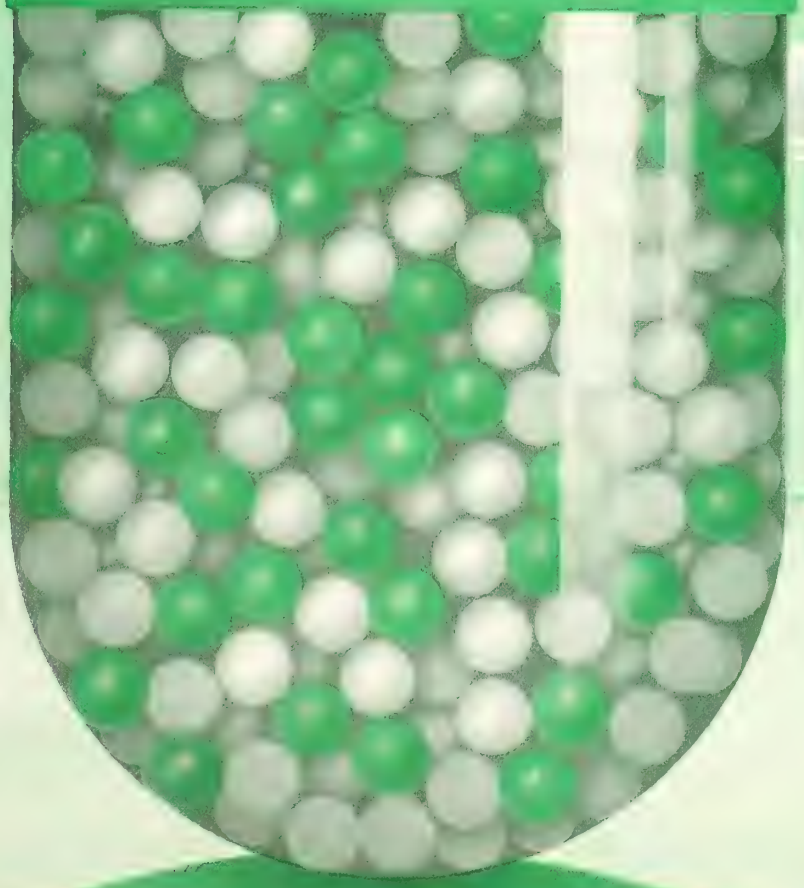

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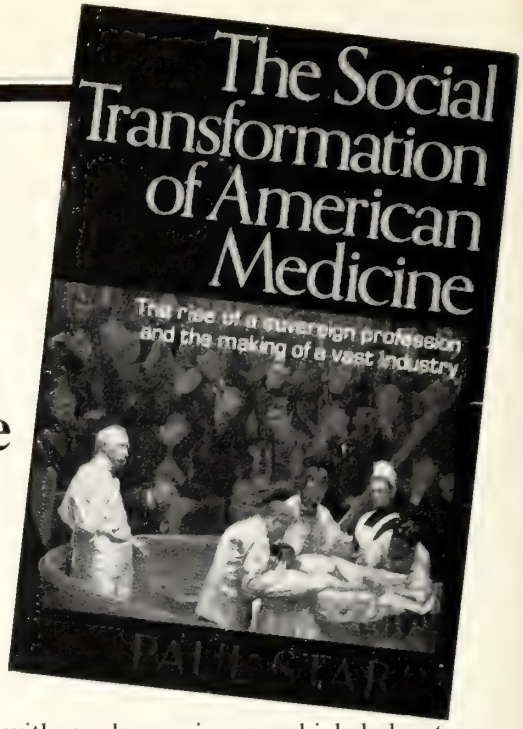


Medicine Meets the Marketplace

Charles E. Davis, Jr., MD, *Norfolk, Virginia*

The Social Transformation of American Medicine, by Paul Starr. New York, New York, Basic Books, 514 pp, $24.95.

How did the American physician come to embody such omnipotent authority? How has this authority remained isolated for so long from the economic and sociologic forces affecting all other elements of our society? Who are the invaders now threatening the doctor's position, autonomy and income?

All this and more, much more, is chronicled in this mature, scholarly history of the American medical profession, told with wit and high drama by a brilliant young associate professor of sociology at Harvard. It is as impeccably documented and referenced as a first-class scientific article. Its scope is encyclopedic; I was thunderstruck by how little I had known about the evolution of my profession. Each page is chock full of facts, yet such is Starr's talent as a writer that his vast array of material flows smoothly and logically.

In his preface Starr says, "My hope is that this historical analysis may help to illuminate our present predicament." That it most certainly does. When you've finished this book, you will know exactly what is happening to medicine today and why it is happening.

The author leads off with "The Social Origins of Professional Sovereignty." This is an erudite appraisal of authority—what it means and how it works. Basically, authority is power, and the establishment of power enabled medicine to become a sovereign profession with more authority than any other, even the law and the clergy. Medicine has close bonds with modern science, which helps to confer preferred status and enhanced credibility. Doctors, says Starr, "serve as intermediaries between science and private experience, interpreting personal troubles in the abstract language of scientific knowledge."

But the physician's authority does not spring from the scientific connection alone. It developed over generations as an admixture of patient dependency, the physician's distinctive education, and the profession's ingrained Hippocratic-like code. Starr calls it "cultural authority." Other professions lack this potent ingredient.

Other factors, including good luck, helped the medical profession establish dominance, eliminate challenges, and gradually gain control of all facets of medicine. Physician monopoly of the commercial aspects of the health field was virtually complete from 1920-1970, and since authority eventually translates into money, medicine became the highest paid of all occupations. Starr notes that "many doctors before 1900 could hardly scrape together a respectable living." What a transition to the medical capitalist of today!

Engineering medicine's rise to power was the American Medical Association, and Starr's careful history of the AMA is one of the most engrossing sections in his book. From its founding in 1846, the AMA's directions and methods have frequently been questioned, often by its own members, but it has consistently defended medicine, as this book makes abundantly clear, and its aggressive lobbying to forestall legislation considered inimical to the profession has been amazingly successful.

Dr. Davis is a Norfolk surgeon and a Past President of The Medical Society of Virginia.

The AMA was, indeed, the instrument by which medicine was welded into a coherent profession. Prior to 1850 there were regular physicians, who were in general disrepute, and there were lay healers. Then sects began springing up, such as the Thomsonians, who believed in botanic medicine. The Eclectics and the Homeopaths came along and gradually forced a merger with medicine. (Osteopathy, chiropractice and Christian Science, all of which came later, have maintained their independence.) Physician unity finally became a reality when, in one ten-year period from 1900 to 1910, AMA memberships increased from 8,000 to 70,000, which was about one-half the physicians in the country. Starr's vivid portrayal of the AMA's history leaves no doubt that without its sustained efforts, American medicine as we know it would never have happened.

By 1920, when American medicine was poised to assume its leadership role in all matters pertaining to health, a sister organization whose origins and development closely paralleled that of medicine was also maturing. This was the American hospital. No part of this book is more fascinating than the chapter titled "The Reconstitution of the American Hospital," in which the escalation of the American hospital's status and public acceptance is described. As Starr puts it, "The American hospital moved from the periphery to the center of medical education and medical practice."

The American hospital's metamorphosis from the loathesome almshouse of the 17th Century to today's sparkling glass and steel structure bristling with electronic gadgets is little short of a miracle. And nothing has so accentuated the dominant role of the American physician as the modern American hospital. The hospital has been the physician's free workshop. Because the physician brought in the patients, to the physician fell the responsibility for formulating hospital policy and establishing rules and regulations. Today, however, great changes are transpiring. Hospital administrators are taking over that authority.

Public Health figured in the struggle for medical autonomy but was bested by private practice. From the beginning, Public Health's habit of establishing free dispensaries and clinics came into conflict with local practitioners, who viewed this as a threat to their income (the pocketbook nerve!) and who let Public Health know that a very definite distinction existed between preventive and curative medicine. On the other hand, the doctors were delighted to yield to Public Health such unremunerative areas as tuberculosis, venereal disease and leprosy. Public Health got the message and slipped into a secondary role in the scheme of things.

The section titled "The Birth of the Blues" is an especially exciting section of this book and will be of unusual interest to Virginia physicians, who are daily involved with this prepayment plan emanating from Richmond and Roanoke. There is general agreement that the Blue Cross concept originated at Baylor University Hospital in Dallas, Texas, in 1929, when 1,500 school teachers were covered in a "single plan," involving only hospital costs, at $6 per person.

Organized medicine took a dim view of such developments. In Los Angeles, in the same year that Blue Cross was launched, Dr. Donald Ross and Dr. H. Clifford Loos agreed to provide 2,000 employees of the city's water and power departments with hospital as well as medical care for about $2 per month and were expelled from their local medical society. Many years later I heard Dr. Ross, a distinguished surgeon and gentleman then in his 70s, describe the early abuse heaped upon him and his partner by their medical colleagues.

In Virginia, six years after the Baylor plan, similar insurance programs originated in Richmond and Norfolk almost simultaneously. And in the early 1940s, as former house officers at the University of Virginia Hospital recall, a prepayment arrangement for teachers was started there; this was the "teachers' preventorium," limited to J ward. Subscribing teachers could be admitted to that floor and were covered for hospital and medical care; their attending physicians were assigned by the various departments.

The AMA early on opposed all prepayment plans as infringements of physician prerogative but later relented and gave approval if the programs were controlled by physicians. Of course, as Starr recounts, the AMA—and most physicians—bitterly opposed Medicare, too, when it came along in the 1960s, and look what a bonanza that became!

In his last chapter, titled "The Coming of the Corporation," Starr describes the development of the vast industry now enveloping and changing medicine, and he projects some possible long-range effects of its growth.

Heretofore we have looked on private enterprise as our friend. No more. Medicine is now itself big business second only to the Pentagon, and private enterprise is invading previously sacrosanct domains, changing traditional ways of practice. A lingering doctor-patient relationship is the only

thing that helps sustain the physician in this new medical pattern. He is necessary for the admission of patients to a facility and therefore still holds a key to the system, which the administrators of the new programs and facilities reluctantly appreciate, but as far as dictating policy, as in the past, he is impotent. It is, Starr says, "the end of a mandate."

Starr envisions progressive increase in corporate dominance. He describes the infinite variety of medical businesses now proliferating. All are in competition. As in other types of commerce, buy-outs occur and mergers are frequent. Both patient and doctor are enmeshed in a strange and different world. Many patients will initially refuse to break long-standing ties with their own doctors, but the exigency of practicality may eventually force them to.

We doctors are now struggling frantically for identification somewhere within this new setting, which we do not even fully comprehend. Everywhere there is evidence that our authority is passé. Like it or not, the guru has been replaced! Or, as the author so aptly puts it, "Medicine has overdrawn its credit."

In his book Starr predicts marked modification of doctor behavior. Thinking processes must be altered to accommodate the new order of things. Doctors must now study "corporate socialization" as well as worry about the medical needs of their patients. Directives originate with the company and are paramount. If the physician doesn't develop a "marketing mentality," he may find he doesn't fit into corporate plans and, thanks to the doctor glut, he may be easily replaced.

Medicine has long had an almost paranoid fear of government socialization, while extolling the virtues of private enterprise. Now the private sector is hoisting us by our own petard.

THERE are lessons to be learned from this book, important lessons. For instance, we physicians like to consider our unparalleled earning capacity as a spinoff of hard work, long education and compassionate dedication, but the history of American medicine makes plain, in plenty of places, that we doctors have been more preoccupied with our incomes than we've been willing to admit. As just one instance, see the renal dialysis story as described by Starr. No better example could be found of the political leverage that can be generated by a small segment of medicine when profit is the crucial motivating factor. For many doctors, this will be a horror story.

Because the compass of this book is, for all its detail, so extraordinary, I was surprised not to find a discussion of the malpractice crisis. I wrote to the author about this and received promptly in return a gracious communication. He sent a copy of an essay he wrote in the late 1970s on the malpractice crisis, and in the accompanying letter he said, "Perhaps you are right that I should have discussed malpractice in the book. I cannot really explain the omission, except to say that an adequate discussion would have led me a bit afield (i.e., the contingency fee system for lawyers). But I suppose that's a weak excuse."

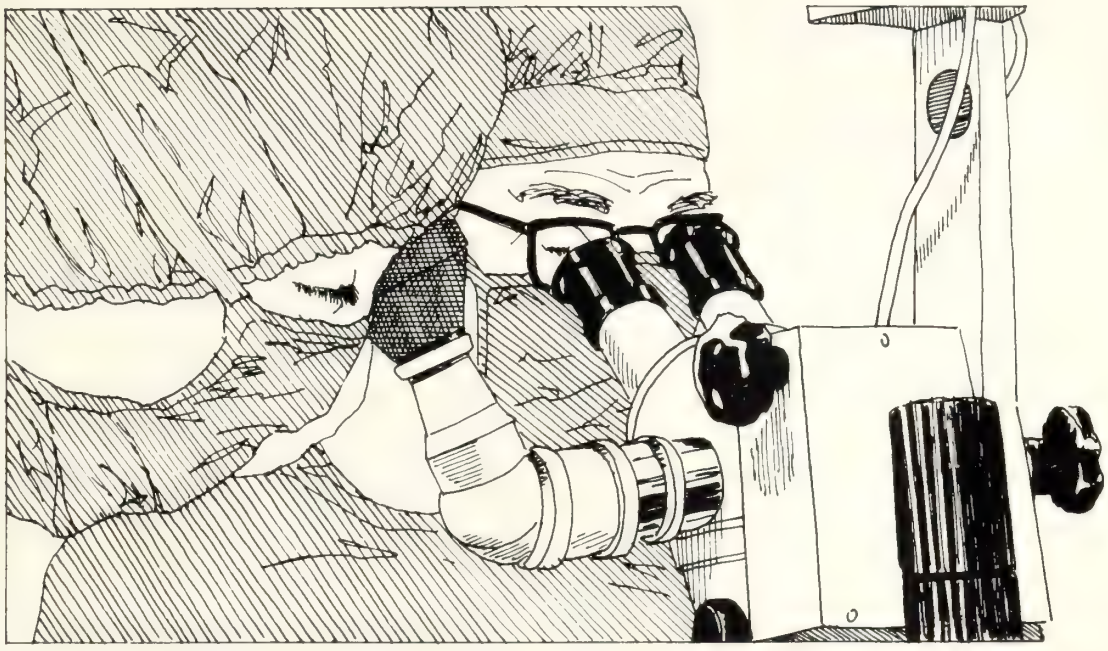
On completion of this book, the physician-reader will certainly feel, as I did, that never before has he or she known so much about the profession of medicine—its origins, its ascendancy, and now, it appears, its decline. An interchangeable title could well be "The Rise and Fall of the American Doctor." Ironically, it is the immutable law of the marketplace that is deposing us. And the doctor-reader will come to the uncomfortable conclusion that he himself has significantly contributed to his own downfall.

The change will entail considerable readjustment by the private practitioner, not the least of which is emotional. Adaptation will surely be easiest for the recent medical graduate, inexperienced as he is with the joys of total independence in clinical practice. He, too, though, will suffer from the continuing erosion of professional prestige. The restrictions limiting our ability to determine such diverse factors as salary, vacations, insurance and retirement will be traumatic, indoctrinated as we are in the habit of exercising complete professional control and personal autonomy.

Heretofore the promised independence and its related benefits have been a major factor in the selection of medicine as a profession. The new format rapidly evolving will surely have staggering effects on the doctor's life and his image. Dr. Starr's book presents no conjecture as to when the current trend will end, but he strongly suspects that "private regulation and corporate planning . . . determined by the rate of return on investments" is the future towards which American medicine is headed.

And this doesn't bode well for the private doctor.

Editors' Note: *You may order* The Social Transformation of American Medicine *by calling Basic Books at 1-800-638-3030 (toll free) with your American Express, MasterCard or Visa number. There is no handling/postage charge.*



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MEETINGS

December 10
Anxiety and Depression in Office Patients (Eastern State Hospital/Taylor Manor Hospital), *Williamsburg*. 6 Cat. I credits. Fee: $50. Mary Ann Ayd, PO Box 16315, Baltimore MD 21210, (301) 435-6562.

January 7-17
Medical Updates V: A Review of Recent Advances in Medicine (East Tennessee State University), *Vail, Colorado*. Sue Hutchinson, MPH, Office of CME, Johnson City TN 37614, (615) 928-6426, Ext. 204.

January 7
Regional Meeting, American College of Physicians, *Washington DC*. Wesley M. Oler, MD, 4800 Van Ness St., NW, Washington DC 20016.

January 7-13
Review of Recent Advances in Medicine (East Tennessee State University), *Park City, Utah*. Fee: $200. Susan Hutchinson, Dept. of CME, Johnson City TN, 37614, (615) 928-6426.

January 19-21
Annual Meeting, Neurosurgical Society of the Virginias, *Williamsburg*. Robert P. Singer, MD, 1651 Parham Rd., Richmond VA 23229.

January 19
Controversies in the Diagnosis and Early Management of Breast and Colon Cancer (East Tennessee State University), *Johnson City, Tennessee*. Sue Hutchinson, MPH, Office of CME, Johnson City TN 37614, (615) 928-6426.

January 29-February 3
Neuroimaging Update (University of Maryland), *Cancun, Mexico*. Program of CME, 10 S. Pine St., Baltimore MD 21201, (301) 528-3956.

January 30-February 2
The Alton D. Brashear Postgraduate Course in Head and Neck Anatomy (Department of Anatomy, Medical College of Virginia), *Richmond*. 40 credit hrs. Fee: $350, MDs; $200, residents. Hugo R. Seibel, PhD, Box 709, MCV Station, Richmond VA 23298.

February 3-5
Winter Retreat: Update on Pulmonary Disorders (Medical College of Virginia), *Wintergreen*. 14¼ Cat. I credits. Fee: $250, MDs; $135, residents. Sheri Rosner, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

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February 10-12
Therapeutic Update on Skin Diseases (Medical College of Virginia), *The Homestead*. 15¼ Cat. I credits. Mrs. Randy Casey, Office of CME, Box 48, MCV Station, Richmond VA 23298, (803) 786-0494.

February 13-17
Clinical Approach to Disturbances of Fluid, Electrolyte and Acid-Base Balance (American College of Physicians), *Vail, Colorado*. Registrar, ACP, 4200 Pine St., Philadelphia PA 19104.

February 14-16
Scientific Assembly: Orthopedic Emergencies (Virginia Chapter, American College of Emergency Physicians), *Wintergreen*. Gwen E. Messler, PO Box 317, Highland Springs VA 23075.

February 23-24
Pediatric Trends (Johns Hopkins University), *Baltimore*. Fee: $450. Edward M. Sills, MD, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-5880.

February 24-26
Annual Meeting, Virginia Chapter, American Academy of Pediatrics and Virginia Pediatric Society, *Williamsburg*. Delosa A. Young, MD, 8316 Traford Le., Springfield VA 22152.

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A Message from the Chairman of the Physician Insurance Committee The Virginia Insurance Reciprocal

I take pleasure in commenting upon the success of the Physician Insurance Program of The Virginia Insurance Reciprocal.

For as long as most physicians in Virginia can remember, we had little choice in the selection of a professional liability insurance company. Many physicians, including myself, felt that this non-competitive environment was not in the best interest of physicians. With this thought in mind, I personally was very enthusiastic about the Reciprocal's entrance into the area of physician professional liability insurance coverage.

The success of the first two years of operation for the Physician Insurance Program is evidence that this enthusiasm has been shared by many physicians and surgeons in all specialties throughout the Commonwealth. The success of this program can be attributed to the financial soundness of the Reciprocal, the emphasis on physician risk management, the professionalism of Reciprocal staff, and, most importantly, to the ability of physicians insured to participate in the operation of the program. As Chairman of the Physician Insurance Committee, I have witnessed first hand the active involvement of Reciprocal physicians in issues and decisions involving claims, underwriting, risk management, and marketing. With such involvement, the Physician Insurance Program is truly a program dedicated to the practicing physician.

No insurance company can afford to become complacent about the many issues affecting physicians today. As Chairman of the Physician Insurance Committee, I am committed to the continued involvement of the Reciprocal's insured physicians in the operation of their insurance program.

Ronald K Davis

Ronald K. Davis, M.D.
Chairman
Physician Insurance Committee
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HOUSE OF DELEGATES CHOOSES LEADERSHIP, CHARTS POLICY

Keeping a sharp eye on such shoals as professional liability, informed consent for breast cancer, and the merchandising of human organs, the House of Delegates set The Medical Society of Virginia on course for the coming year in its annual session at the Homestead in November.

The delegates also selected new leadership. Dr. C. Barrie Cook took the helm as President; to read of the distinguished experience he brings to the presidency, see page 723 of this issue.

Dr. Harry C. Kuykendall was chosen President Elect. The three new vice presidents are Dr. Anthony J. Munoz, Dr. Robert C. Green, Jr., and Dr. Darrell K. Gilliam. Re-elected were Dr. Richard L. Fields, Speaker of the House; Dr. William H. Barney, Vice Speaker; and James L. Moore, Jr., Executive Vice President.

Two new councilors were elected, Dr. William W. Regan and Dr. John A. Owen, Jr., and one vice councilor, Dr. Ashley Futral, Jr. Dr. William J. Hagood, Jr., became an AMA delegate, while Dr. Harold L. Williams joined the contingent of alternates, and Speaker Fields was made a member of the AMA delgation.

For a complete roster of MSV officers 1983-1984, see page 681.

The heavy work had been mapped out by the reference committees. Reference Committee One, with Dr. Charles C. Ashby as chairman had considered actions having to do with education and interprofessional relations. Internal affairs were the charge of Reference Committee Three under Dr. C. M. Kinloch Nelson's leadership. Issues relating to legislation and regulation had been considered by Reference Committee Two, chaired by Dr. Carol S. Shapiro. Significantly, to this last committee fell 14 resolutions, while the other two together fielded only six.

Here are major moves the delegates made. A report of all their decisions will appear in Virginia Medical's January issue, along with a portfolio of pictures taken at the meeting.

TO TAX OR NOT TO TAX?

The tax-exempt status of prepaid health plans was the subject of lively debate in reference committee. At issue: a 2.75% premium tax that insurance firms are required to pay. A recommendation from the Legislative Committee called for the General Assembly to remove the exemption, and the reference committee finally voted to adopt it.

But in the House, after Past President W. Leonard Weyl rose to adjure the physicians to steer clear of the controversy, the delegates rejected the recommended position. Instead, they approved a substitute motion calling for the General Assembly to study "the effect of legislating tax-exempt status for insurance carriers, prepaid health care plans, and other private third-party payers, focusing on competition and health care costs."

CONSENT AND CANCER

Amid hot tempers and headlines, a proposal to mandate informed consent for breast cancer patients lost in last year's General Assembly, but proponents are said to be tuning up for a rerun in 1984. The delegates reiterated the Society's opposition of last year to any legislation that would require doctors to tell newly diagnosed breast cancer patients of treatment alternatives.

Separately, the House asked the

Society to join the American Cancer Society in sponsoring education programs for physicians on options for breast cancer therapy.

TRANSPLANTS AT RETAIL?

Sales of human organs for transplant purposes should be prohibited by state law in Virginia. That was the unequivocal message of a resolution passed swiftly and unanimously.

LIABILITY EASEMENTS

Medical malpractice entered the delegates' deliberations through three resolutions. All were adopted. They asked Virginia's legislators to study

1. modification of the colateral source rule so that payments to a plaintiff by an independent source could be introduced as evidence in liability litigation;

2. institution of greater equality in the awards system, which now confers sums four times larger for injuries allegedly due to medical malpractice than for similar injuries sustained in auto accidents.

3. placement of a "reasonable" cap on awards for pain and suffering in medical malpractice cases because the resulting higher insurance premiums contribute to rising costs.

AUTO SAFETY

Seat restraints for infants in motor vehicles was the object of legislation that passed the 1983 General Assembly with MSV support. This year, the House decreed, the Society is to support a law that would require everyone, young and old alike, to be secured by safety devices when riding in autos of post-1968 manufacture.

MD REIMBURSEMENT

Three actions dealing with reimbursement were endorsed by the delegates. One asked MSV support of legislation "which forbids prepaid medical and surgical plans from prohibiting a subscriber from assigning right to payment for covered services to the physician of the subscriber's choice."

Another, referring to Report D of the AMA Council on Medical Service, asked the AMA to "seriously consider recommending that third parties change to an indemnity system of payment for physician services, i.e., paying a set amount for services rather than some proportion of the UCR charge."

The House also adopted a resolution asking Medicaid officials for concurrent review of claims for reimbursement rather than the retrospective system now operative.

CERTIFICATES FOR TWO

In separate recommendations, the Legislative Committee asked for certification of athletic trainers and respiratory therapists. The reference committee approved and so, too, did the House.

EDUCATING PATIENTS

The American Medical Association's Patient Medication Instruction Forms, or PMIs, generated a resolution pledging The Medical Society of Virginia to take "an active and aggressive position in support of the use of PMIs by Virginia physicians." The delegates unanimously approved.

TREATING PATIENTS

The House adopted a recommendation in support of legislation requiring immunization against rubella for all students born after 1963 matriculating for "post-scholastic" studies at two- and four-year colleges and trade and professional schools in Virginia.

ON DEATH AND DYING

A change in Virginia's Natural Death Act, passed with Society support by last year's General Assembly, was requested in action by the delegates. They asked for exemption for the situation "where an incompetent patient without a declaration suddenly becomes terminally ill and no family is available to make a decision about the withholding of life-prolonging procedures."

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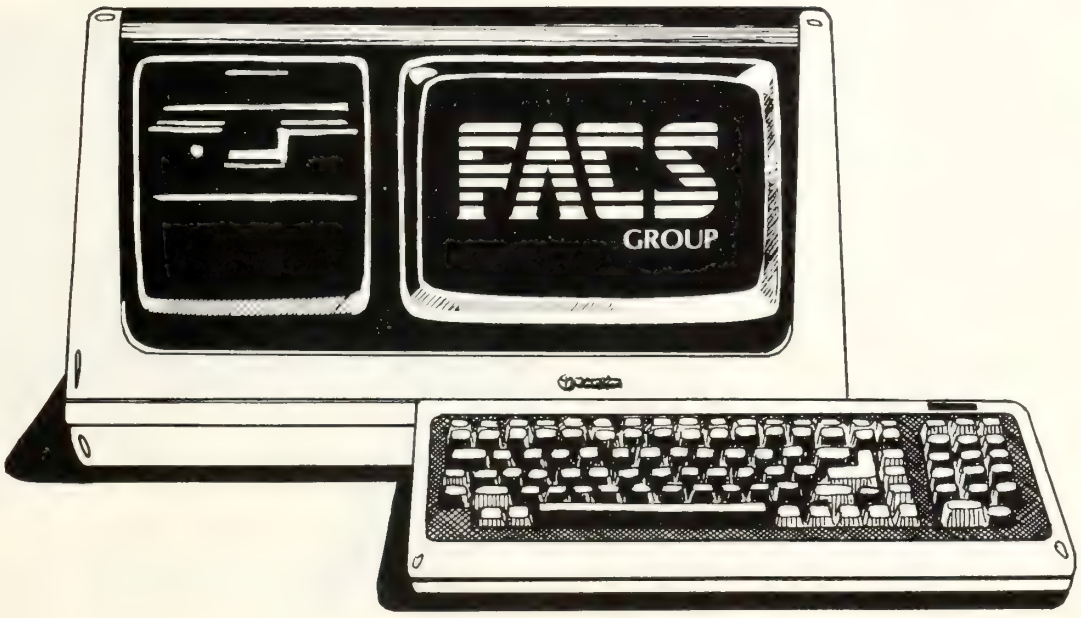

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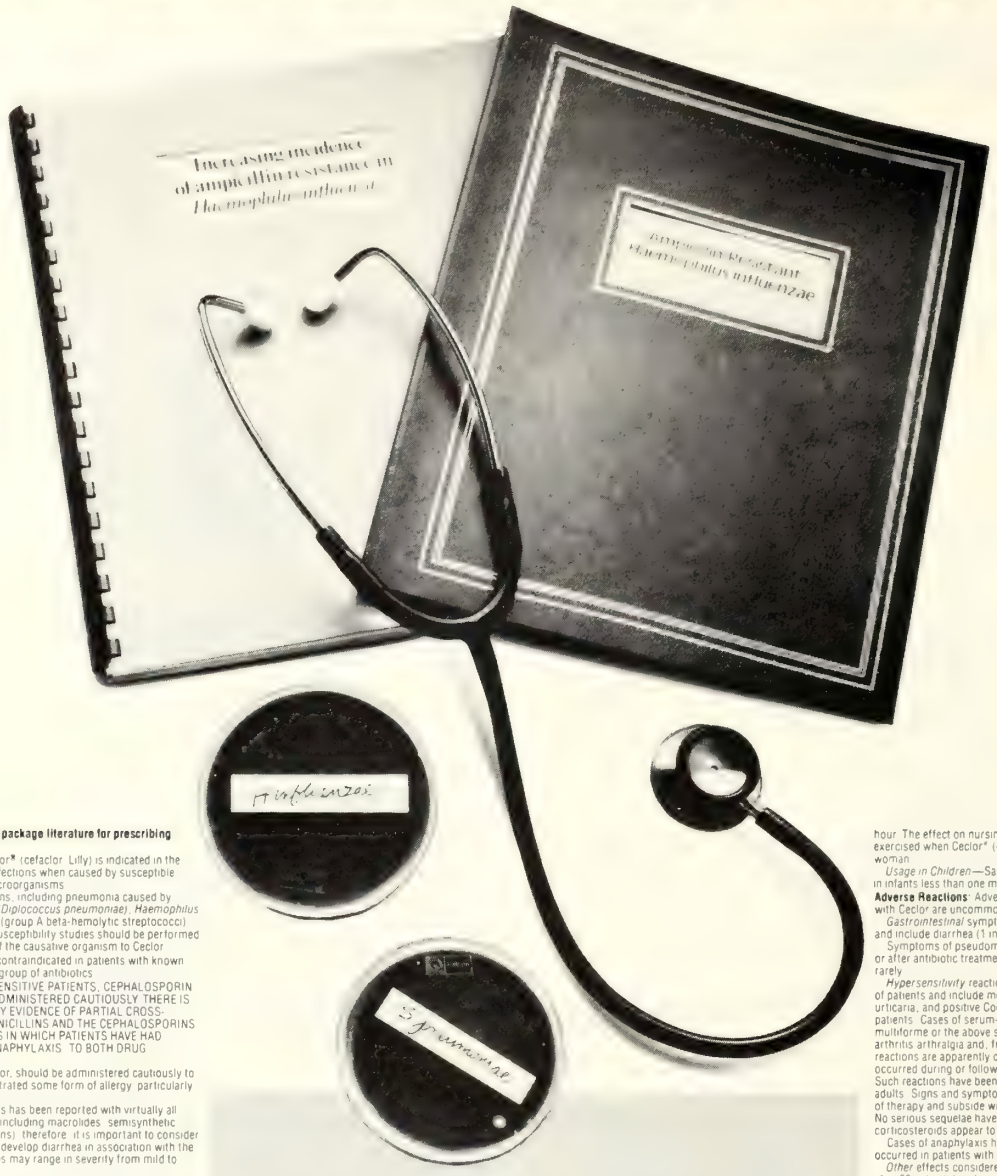

Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

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Brief Summary Consult the package literature for prescribing information

Indications and Usage. Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings. IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions. *General Precautions*—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor® (cefaclor, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below.

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

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*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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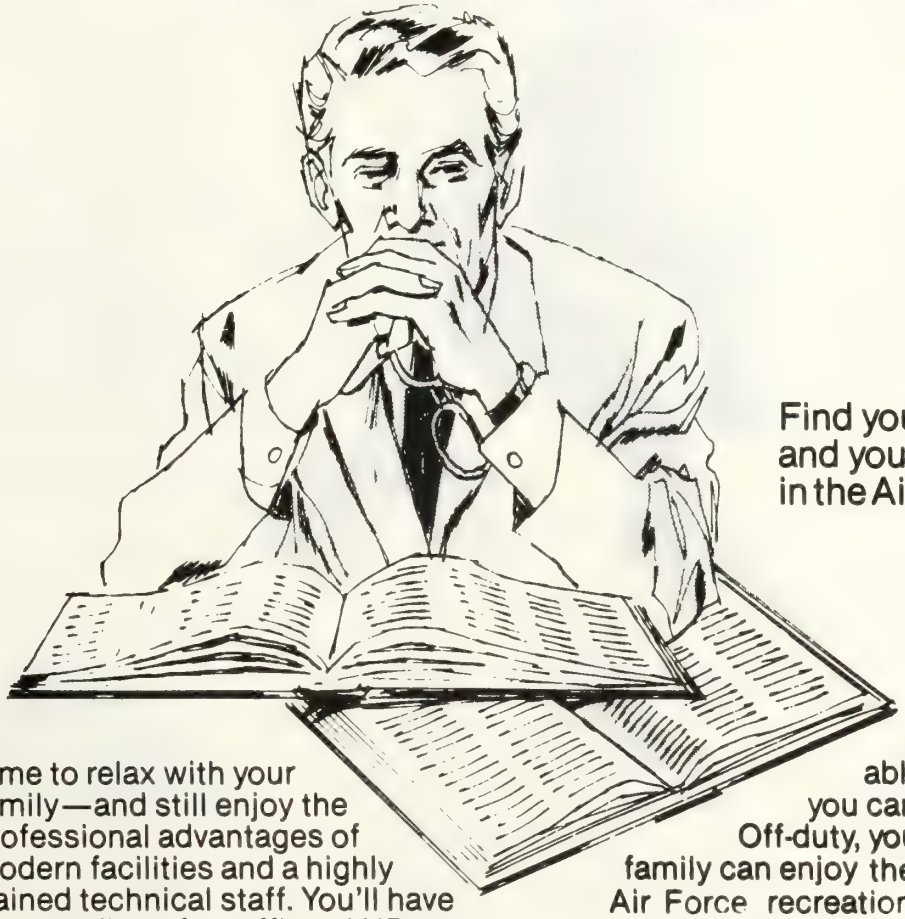

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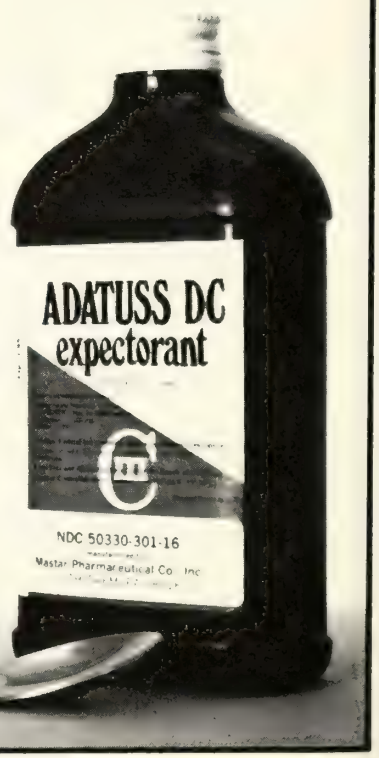

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C1-Inhibitor Deficiency (Hereditary Angioedema): Case Report and Review

Joseph J. Burge, MD, *Charlottesville, Virginia*

Often misdiagnosed as an allergy, hereditary angioedema, or C1-inhibitor deficiency, is curable but can threaten the patient's life if appropriate therapy is delayed. A case report and review describe successful treatment of this unusual disease.

Case Report

S.R., a 32-year-old white female schoolteacher from Roanoke, Virginia, was referred to the University of Virginia Lupus Clinic for evaluation of recurrent facial and extremity edema. She first noted marked swelling of her right leg following a trivial ankle injury at age 14; similar episodes recurred, associated with minor trauma. She also noted the onset of frequent bouts of severe crampy abdominal pain during her teenage years lasting 1-3 days. These attacks were associated with constipation and anorexia, they occurred spontaneously, and they were unrelated to episodes of skin edema. She began a three-year course of allergy desensitization at age 17 but without benefit. The patient was again evaluated at age 18 and her complaints attributed to excess nervousness. A belladonna/phenobarbital medication was prescribed without response.

Her first episode of facial swelling occurred seven years ago at age 25 after biting her lip. She also reported a 12-hour episode of throat swelling progressing to respiratory distress following wisdom teeth extractions. Over the past three years her attacks of facial and extremity edema increased in frequency and severity and at the time of evaluation were occurring weekly. Her most recent attack followed a slight blow to her upper lip from her three-year-old child the day before her referral. These episodes were not seasonal in occurrence and typically followed minor trauma. The affected skin was not tender, discolored or pruritic, and the swelling resolved in 3-5 days without residua. Multiple drugs, including antihistamines, epinephrine and prednisone, had been tried unsuccessfully by a number of physicians. There was no family history of similar complaints.

From the Division of Rheumatology, Department of Internal Medicine, University of Virginia School of Medicine. Address correspondence to Dr. Burge at Box 412, University of Virginia Medical Center, Charlottesville VA 22908.

Submitted 3-23-83.

1. Hands of the patient S.R. during an attack of angioedema. Only the right hand is affected.

The patient was a thin white woman, BP 110/70, T 37°C. The HEENT exam was remarkable for swelling of the right cheek and upper lip without signs of trauma or inflammation. The remainder of the general physical exam was unremarkable.

CBC, platelet count, urinalysis, and ESR were normal. Assays for rheumatoid factor, cryoglobulins, anti-nuclear antibodies (ANA), anti-DNA antibodies, anti-ENA antibodies and immune complexes were all negative. Complement studies included a CH50 of 15 U/ml (N = 34-48), C3 58 mg/dl (N = 55-120), and C4 2.5 mg/dl (N = 20-50). Serum protein electrophoresis (SPEP) revealed a relative decrease in alpha-2 globulins. C1 esterase inhibitor by radial immunodiffusion was 5% of normal.

A diagnosis of hereditary angioedema was made and treatment was begun with stanozolol 2 mgs po qd. She was told to postpone planned dental work for a week and further instructed to take 4 mgs stanozolol qid for four days prior to any dental work or surgery. The patient was warned of possible mild masculinizing side effects of her drug therapy and advised to discontinue its use in the event of pregnancy. She has had no attacks of angioedema over the three-month period since beginning therapy and even underwent a dental procedure without complication.

Discussion

Hereditary angioedema (HAE) is a clinical syndrome occurring in young adults and characterized by recurrent attacks of subcutaneous swelling, abdominal pain and laryngeal edema, and associated with decreased functional levels of the serum protein, C1-inhibitor (C1 INH)[1]. This is probably not the best name for the syndrome because it is not always inherited. Furthermore, angioedema is a very general term; many different pathologic mechanisms can lead to angioedema.

When Osler first described the syndrome in 1888, he referred to it as "hereditary angioneurotic edema". In Osler's time, "neurosis" was used in the sense of nervous system control with "angioneurotic" referring to nervous system control of blood vessels, his theory of the pathogenesis of the syndrome. Now, of course, "neurosis" is used almost solely in its psychological connotation, but unfortu-

nately this change in usage has led in some cases to the misconception that the syndrome is triggered by the individual's psychological state.

Perhaps the best way to refer to this syndrome is as "C1-inhibitor deficiency", which can then be further designated as hereditary or acquired. Our patient has C1-inhibitor deficiency, and her symptoms and mode of presentation of diagnosis are classic for the syndrome.

Signs and Symptoms

The major symptoms associated with C1-inhibitor deficiency are the triad of angioedema, laryngeal edema and abdominal pain. Figure 1 shows an attack of angioedema of the patient's right hand. This is deep subcutaneous edema, not urticaria, which is more superficial. Angioedema is nonpruritic, non-painful, non-pitting and not particularly inflammatory.

The usual sequence of development of angioedema is an episode of minor trauma followed soon afterward by a tingling or tightness under the skin, and then the appearance within one-half to one hour of angioedema, which spreads for a few hours. The angioedema remains for 24-72 hours, then resolves spontaneously.

The patient also had an episode of laryngeal edema following a dental procedure. Dental procedures are known to be particularly dangerous in these patients because of upper airway obstruction, which is the most common cause of death in patients with C1-inhibitor deficiency. The laryngeal edema can also be spontaneous and in these cases, the patients usually feel the edema coming on slowly. They almost always have a voice change and dysphagia before going on to obstruction, so these are warning signs which would lead the physician to elective endotrachial intubation, or tracheostomy.

The patient also manifested the third major symptom of C1-inhibitor deficiency, that is, abdominal pain with nausea and mild diarrhea. This is due to intestinal edema, and a GI series performed during attacks should show the characteristic "thumbprinting" pattern of intestinal wall edema[2]. Colicky pain is a common symptom along with nausea. Diarrhea develops later and is usually mild and watery. The patient is afebrile, has no leukocytosis, no peritoneal signs, only moderate abdominal tenderness, and normal or increased bowel sounds. The diarrhea never contains white cells or blood. Occasionally, a patient may have severe sequestration of fluid in the gut and develop hypotension or even shock, which may resemble true anaphylaxis.

Differential Diagnosis

In regard to differential diagnosis, most often these patients are misdiagnosed as having an "allergy", i.e., immediate hypersensitivity. In fact, there is no element of allergy in this disease. The pathogenic mechanism is different, there is no eosinophilia, and it is unresponsive to the usual allergy treatments, such as epinephrine, steroids or antihistamine.

The other major problem in differential diagnosis is the patient who presents with GI symptoms but no peripheral angioedema. Many of these patients are worked up for what appears to be an acute abdomen and even go to surgery, which can be life-threatening to them if the diagnosis is unsuspected. The GI symptoms most resemble those seen in ischemic bowel disease, such as sickle cell anemia or vasculitic bowel infarcts; however, the patients with C1-inhibitor deficiency are not anemic, are afebrile, and have no WBC or blood in their diarrhea. The patient presenting with low-grade GI symptoms is often labelled as "hysterical".

Laboratory Findings

As for laboratory findings in C1-inhibitor deficiency:

1) The deficiency itself is reflected in 85% of the patients by low serum levels of C1-inhibitor.[3] They are seldom totally deficient of the C1-inhibitor but rather have levels 5%-20% of normal. However, 15% of the patients have *normal* antigenic serum levels of an *abnormal C1-inhibitor molecule* which has decreased or absent *functional* ability. The C1-inhibitor serum assay as routinely performed detects only the *antigenic* levels, so that one might miss this 15% of patients unless one performs a *functional* test of the C1-inhibitor, a much more elaborate test.

2) These patients have low levels of C4 and C2, and, of course, a low CH50 because of the depleted C4 and C2. They have normal C3 levels, however, because other complement control proteins come into play to protect the C3.

3) There may be a relative decrease in alpha-2 globulins (of which C1-inhibitor is a member) on serum protein electrophoresis.

4) The serum C1 levels are important in distinguishing between *hereditary* C1 deficiency and *acquired* C1-inhibitor deficiency. In acquired C1-inhibitor deficiency, the C1-inhibitor has been "used up", if you will, by an excess amount of activated C1.[4] This is usually seen in the setting of hematologic malignancy and monoclonal gammopathies, where there are abnormal antibodies and immune

complexes present which activate C1 and deplete the body's C1-inhibitor supply[5]. In the hereditary cases, C1 level is normal or increased.

5) All of the other laboratory values in these patients are normal, including chemistries, white cell counts, eosinophil counts, hematocrit, sedimentation rate, and number of rheumatological parameters, such as rheumatoid factor, ANA, anti-DNA and immune complex assays.

Course

Attacks of angioedema due to C1-inhibitor deficiency vary dramatically in frequency and duration, even in the same patient. In patients with the hereditary form, the attacks first appear in childhood, are usually manifested by angioedema of an extremity, and frequently are overlooked as mild trauma since they are self-limited. More severe symptoms occur at puberty, and in fact, most patients date the onset of their symptoms to puberty. Men and women are affected equally. Menstruation seems to induce attacks in women (but not necessarily in the genital region); menopause, however, doesn't decrease the frequency of attacks. Birth control pills are known to exacerbate symptoms. A number of patients with C1-inhibitor deficiency have had normal pregnancies, as our patient did; in fact, it appears that attacks actually decrease in frequency during pregnancy. Birth-related trauma also does not seem to induce an attack.

This syndrome often goes undiagnosed for years, for example, in the National Institutes of Health study[1], the average duration of symptoms before diagnosis was 21 years. Since the patient of this report went 18 years without diagnosis, she was typical in this regard. Most cases are of the autosomal dominant hereditary variety; therefore, the patients will have some family history of similar attacks, although this is not always so, as we see in our case. In the NIH study, seven of 30 patients did not have a positive family history of the disease.

Pathogenesis

The C1-inhibitor has been isolated from serum, and antibodies to it are available. It has molecular weight of 100,000, has a very high carbohydrate content and is synthesized in the liver. The normal serum concentration is about 15 mgs per dl which makes it reasonably abundant in serum, and it is an acute-phase reactant, that is, its levels are increased in inflammatory states, as one might expect, since it is a normal control protein.

The C1-inhibitor is an inhibitor of the C1r and C1s subcomponents of C1; it is an inhibitor of the kinin-generating system through its action on Hageman factor and kallikrein; it is an inhibitor of the clotting system through its action on Hageman factor and factor XI; and it is an inhibitor of the fibrinolytic system through its inhibitory action on plasmin.

It normally functions as an inhibitor (control protein) of C1, the first component of the classical complement pathway. The C1 molecule is made up of three subcomponents, C1q, C1r and C1s, and binds in 1:1 fashion to the active enzymatic sites of C1r and C1s, irreversibly inhibiting them. The resulting complexes are then cleared from the body as units. Although there is a great deal known about the C1-inhibitor and its function, there is still no complete explanation of the pathophysiologic events involved in an attack or why the attacks are self-limited.

There are a number of pathologic mechanisms involved in urticaria and angioedema, and there has been a great deal of research into them.[6] The three major factors contributing in varying degrees to angioedema include release from mast cells of inflammatory mediators, such as histamine, leukotrienes, prostaglandins, enzymes, etc; accumulation of inflammatory cells, such as eosinophils and neutrophils, in response to chemotactic factors; and the generation of bradykinin and other kinins, which increase vascular permeability.

Simply stated, one does not see in hereditary angioedema the elevations of plasma histamine characteristic of many of the allergic types of urticaria and angioedema. There is little or no infiltration of the skin with eosinophils or neutrophils, but there *is* kinin-like activity generated during the attack. Thus, the current theories regarding the pathophysiology of angioedema related to C1-inhibitor deficiency attempt to explain the local generation of kinins through self-limitation. Two major theories are that the kinin is generated directly by the complement system or that the kinin is in fact bradykinin from the endogenous kinen-generating system.

In regard to the former theory, the classical pathway of complement is most commonly activated by immune complexes interacting with the first component of complement, C1q component of which binds to the Fc portion of IgG or IgM. This in turn allows the binding of C1r and C1s. C1-inhibitor directly inhibits C1r and C1s. The ordinary function of C1 is to cleave C4 and also C2. The enzymatic site for cleavage is on the C1s molecule. If there is deficiency of C1-inhibitor, this C1s can go unchecked in its cleavage of C4 and C2, and it is felt that a fragment of the C2 molecule that is cleaved,

Table 1. Therapy of C1-Inhibitor Deficiency

I. Long-term prophylaxis
   1. Anti-fibrinolytic agents (epsilon amino-caproic acid; tranexamic acid)
   *2. Modified androgens (stanozolol; danazol)

II. Short-term prophylaxis
   *1. Fresh frozen plasma
   2. Epsilon amino-caproic acid orally
   *3. Modified androgens (stanozolol; danazol)
   4. C1-inhibitor concentrate

III. Acute attacks
   1. Modified androgens (stanozolol; danazol)
   2. Epsilon amino-caproic acid intravenously
   *3. C1-inhibitor concentrate
   *4. Conservative management, i.e., cold application, narcotics for abdominal pain, tracheostomy if necessary

*Treatment of choice

the portion called C2b, is further cleaved in some way in the body into a kinin-like material capable of increasing vascular permeability and of causing the angioedema.

An alternative mechanism involves the kinin-generating system in human blood.[7] A number of the steps of this system are normally inhibited by C1-inhibitor; thus, in its absence, this system would be expected to go uncontrolled and lead to production of large amounts of bradykinin, which in turn can cause angioedema. However, the experimental injection of bradykinin into animals does not totally simulate all the characteristics of the angioedema associated with C1-inhibitor deficiency.

To summarize, it appears that the pathophysiology of angioedema related to the C1-inhibitor involves the generation of a kinin-like substance locally, but details of this kinin generation, the initiating event, and the factors which limit the reaction are still under investigation.

Treatment

As outlined in Table 1, treatment of C1-inhibitor deficiency can be divided into three phases: long-term prophylaxis, short-term prophylaxis, and management of acute attacks. In the past, anti-fibrinolytic agents such as episilon amino-caproic acid (EACA)[8] or an analog, tranexamic acid, were used in long-term prophylaxis. Both C1-inhibitor and these agents inhibit the same enzymes, but the latter have significant toxicity and have been replaced by the use of modified androgenic hormones[9,10].

In 1960, Spaulding reported the use of methyltestosterone in the treatment of hereditary angioedema. He decided to try this on the advice of a pathologist who was doing research on animals regarding histamine release, and had found that methyltestosterone inhibited histamine release. He used the methyltestosterone in humans under the mistaken impression that histamine played a role in hereditary angioedema; this was wrong thinking but resulted in the observation that androgenic hormones do in fact dramatically decrease the number of attacks of hereditary angioedema. All of this was three years *before* it was known that hereditary angioedema was related to C1-inhibitor deficiency. Now we know that androgenic hormones work by increasing the endogenous synthesis of the C1-inhibitor.

Over the years, efforts have been made to find androgenic hormones with less masculinizing effect and more protein anabolic effect. They raise the serum levels of the C1-inhibitor not back to normal but up to at least double or triple the base-line level, which apparently is enough to prevent recurrent attacks. We treated the patient of this report with the anabolic steroid stanozolol (Winstrol®)[11], which has very little masculinizing effect and is lower in cost than another androgenic hormone, danazol (Danocrine®). Although stanozolol completely abolished all attacks in our patient, the more typical effect is to decrease the number of attacks quite dramatically but not totally abolish them.

When we speak of short-term prophylaxis, we mean, for example, in preparation for a dental procedure or surgery. In this respect, before the use of androgenic hormones, two studies showed that fresh frozen plasma was effective as a source of C1-inhibitor in patients with hereditary angioedema for short-term prophylaxis when give 1-2 days before dental procedure or surgery.[12] Unfortunately, in some patients the administration of fresh frozen plasma only supplies more substrate for the disease, that is, it supplies C4 and C2, exacerbating the angioedema. Why this doesn't occur in every patient is not known. It is known that stanozolol in high doses, for example, 4 mgs four times a day over 4-5 days before oral surgery induces the production of endogenous C1 inhibitor. In fact, the stanozolol begins the induction of C1-inhibitor synthesis within 24 hours of starting the medication.

Other alternatives in short-term prophylaxis are the administration of purified C1-inhibitor, but I believe this is best reserved for management of acute attacks. Epsilon amino-caproic acid is no longer used in the short-term prophylaxis.

For acute attacks, stanozolol has been used in high doses in the same way as for short-term

prophylaxis. Since it acts so quickly, it can often abort the attack, especially if started early. Again, fresh frozen plasma is a problem as described above. Some people use fresh frozen plasma and stanozolol to treat this problem. The optimum therapy for an acute attack is injection of purified C1-inhibitor, as demonstrated with dramatic success by two groups, one at the National Institutes of Health[13] and the other in Italy.[14] Unfortunately, purified C1-inhibitor is not readily available, although the American Red Cross plans to make it so in the near future. The other mainstay of the acute attack is endotracheal intubation, as mentioned earlier. Again, epinephrine, antihistamines and corticosteroids do not affect the acute attack, and no delay should be incurred by attempting these drugs.

One thing that can be helpful in an acute attack of laryngeal edema at home is for the patient to swallow ice chips and keep the area as cold as possible, until the hospital is reached. This is known to decrease the rate of progress of the attack.

Conclusion

The patient reported here suffered for 18 years with a rare, potentially life-threatening but treatable disease. Her case dramatizes the importance of increased physician awareness regarding C1-inhibitor deficiency.

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Radiation Therapy for Renal Transplant Rejection Reactions. William J. Peeples, MD, Duane G. Wombolt, MD, Anas M. El-Mahdi, MD, and Cornelius I. C. Turalba, MD, *Norfolk*.

Forty-four renal transplant patients were given radiation therapy for severe rejection phenomena. The 29 patients who had only one course of irradiation had a 52.3% successful function rate. Fifteen patients received from two to four courses of irradiation with an ultimate 60% rate of sustained function. Fifty patients who received only steroid and other medical management but no irradiation had a 60% rate of successful renal function. In the irradiation group, no patient whose creatinine level did not respond to radiation therapy maintained a functioning kidney. The data indicate that the overall successful function rate is maintained by radiation therapy in patients with severe allograft rejection phenomena, South Med J 75:30–32, 1982.

Screening Hospital Employees for Colorectal Cancer

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At two large Virginia hospitals, employee response to colorectal screening programs was disappointing, both in number of slides returned and percentage of participants over 50 years of age. Out of this experience, the authors offer recommendations for enhancing the effectiveness of such programs.

As major sources of employment in many communities, hospitals can play an active role in health maintenance and in early detection of potentially serious illnesses, specifically through screening programs.

The ideal method for colorectal cancer screening has yet to be developed although fecal occult blood testing with hemoccult slides has been the method most frequently used in large programs.[1–3] Evidence from controlled studies suggests that early detection of colorectal cancer is beneficial in terms of finding lesions localized to the bowel wall[4–6]; survival is improved in comparison to patients with lesions that have spread to regional nodes or distant sites.[5,6]

The response to any screening program depends on several factors.[7] The level of sophistication of the population being screened, the degree of health awareness, and the overall perception and attitude towards the disorder being screened are critical determinants contributing to a successful response. Unfortunately, these are difficult to influence. Crucial factors more readily controlled include the organization of the program and the extent to which the program is publicized.

We designed programs at two hospitals in central and western Virginia to assess the response of hospital employees to colorectal cancer screening. To enhance employee participation the programs were carefully organized and widely advertised. The response to these programs is described in this report.

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Materials and Methods

During the winter of 1980-1981 colorectal cancer screening for hospital employees was performed at the University of Virginia and Roanoke Memorial Hospitals. The former is the major teaching hospital of the University of Virginia School of Medicine, a state-supported institution, and has 2600 hospital employees; about 20% (520) of them are more than 50 years of age. Roanoke Memorial Hospital is the largest non-profit community hospital in the state and is affiliated with the University of Virginia Medical School. It has 2,000 hospital employees and about 20% (400) of them are more than 50 years of age.

Both screening programs utilized the group meeting method[8] for slide distribution but differed in a few respects. The University of Virginia Hospital colorectal cancer screening program was introduced by a memorandum distributed by mail to all employees describing the nature and purpose of the program and stating that the program was to be administered through the Employee Health Department. Factors known to increase the risk of colorectal cancer were specified in the memorandum (age >50 years, personal and family history of colorectal cancer or polyps). This was followed by an article in the hospital publication emphasizing the same points. Posters encouraging participation were placed in areas frequented by employees. During the next three months, the head nurse of the Employee Health Department regularly attended scheduled meetings of hospital departments to explain the screening program and encourage high-risk individuals to participate. Details regarding dietary manipulation and preparation of hemoccult slides were provided. Employees could obtain slides in the Employee Health Department after the meeting; their names were registered in a log.

In the Roanoke Memorial Hospital colorectal cancer screening program, a memorandum describing the program was distributed to all employees with their paychecks. Two days later the program director conducted two information sessions with interested employees. Audiovisual aids were used to explain the purpose of the program and to provide the details related to hemoccult slide preparation. The names of the employees taking slides were not registered.

In neither screening program were those who took slides but failed to return them further encouraged to participate.

Table 1. Colorectal Screening Programs for Employees at Two Hospitals

	Uni. of Virginia Hospital	Roanoke Memorial Hospital
Total number employees	2600	2000
Target employees (>50 years)	520	400
Employees contacted, all ages	550	173
Employees obtaining slides	46	154
% of contacts	8.4%	89.0%
% of target	8.9%	38.5%
Employees returning slides	31	30
% of contacts	67.4%	19.5%
% of target	6.0%	7.5%

Results

In the University of Virginia Hospital program, 550 employees of all ages were informed of the colorectal cancer screening program at regular department meetings, and 46 (8.4%) subsequently obtained hemoccult slides. Although 31 returned them, for a high return rate of 67.4% (Table 1). No participant was found to have occult blood in the stool.

At Roanoke Memorial Hospital, 173 individuals attended the information sessions, 154 (89% of attendees) took slides, and 30 returned them for a return rate of 19.5%. These 30 persons represent 7.5% of employees over 50 years of age. Five employees had one or more slides positive for occult blood. Investigation did not reveal abnormalities in these employees, yielding a 16.5% false-positive rate.

Discussion

The response of the hospital employees to these programs was poor in spite of widespread publicity and careful organization. Negative attitudes towards screening in general, and colorectal cancer in particular, may have played prominent roles in the disappointing response.

Although the group-meeting method was utilized for slide distribution at both hospitals, variations in method led to considerable differences in the results, for example, the poor response rate at the University of Virginia Hospital, despite extensive publicity and the major efforts of the head nurse. A factor to be considered in interpreting this poor response rate is that employees were contacted in non-selective fashion at meetings not expressly held to discuss the screening program. Hence many persons at these meetings were not targeted as part of the population to be screened. The results are also disappointing in another context, that of screening the estimated 520 employees over 50

years of age; even if all 46 employees who took slides had been in this age group, the success rate for this older group would have been only 8.9%.

Since hemoccult slides were not distributed at the personal contact meetings, those who obtained these slides from the Employee Health Department were specifically motivated to do so, as reflected by the high return rate of 67.4%, but the 31 individuals returning the slides represent only 5.6% of the 550 employees contacted and 6% of the 520 employees older than 50.

In the program at Roanoke Memorial Hospital, the information sessions were held shortly after the program was advertised and were expressly for employees interested in participating in the screening program; accordingly, 89.5% of employees attending these sessions took hemoccult slides. Only 19.5% of these persons returned the slides, however. This may reflect a sense of obligation after attending the information sessions rather than an interest in the program itself. A disappointing 7.5% of persons over 50 years of age returned slides.

Recommendations concernng restriction of red meat and ingestion of a high-fiber diet were not emphasized in the Roanoke Memorial Hospital screening program, a factor that may account for the 16.5% false-positive rate.

The response of hospital employees to colorectal cancer detection may be improved by modifying our methods. We recommend using the group-meeting method and introducing the program with a memorandum to all employees accompanying their paycheck. The memorandum should state simply the goals of the program and should identify employees who would benefit most by participating. Intensive publicity is necessary.

One day should be designated for voluntary information sessions concerning the program and for slide dissemination. These ssssions should be held shortly after the program is advertised and at different times during the day to allow as many employees as possible to attend. During these sessions, the problem of colorectal cancer should be reviewed and the simplicity of the hemoccult slide test emphasized.

Slides should be distributed at these meetings to those employees who say they intend to return them. The names of those receiving slides should be recorded in a log, and they should be contacted if the slides are not returned within a reasonable period. Employees found to have one or more positive slides should be referred for a complete evaluation.

By incorporating these recommendations we believe the response to colorectal cancer screening programs for hospital employees can be enhanced.

Summary

Our experience with these two programs for hospital-employee screening has led us to conclude that basic attitudes towards health awareness and early detection of disease can diminish the effectiveness of screening programs, even if these programs are well-organized and well-publicized. Factors that determine an individual's willingness to comply with a screening program are at least as important as how well the program is administered. Understanding and influencing these factors is a difficult task, requiring further evaluation.[7]

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Interstitial Pulmonary Edema Due to Hydrochlorothiazide: Case Report

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THE PURPOSE of this paper is to point out that an allergic reaction to hydrochlorothiazide must be considered in the radiographic differential diagnosis of acute pulmonary edema particularly with an interstitial type of pattern.

Case Report

The patient, a 50-year-old white female, was admitted to Culpeper Memorial Hospital on 7 February 1980 because of an acute onset of wheezing and cough approximately one hour after ingestion of one 50-mg tablet of hydrochlorothiazide for the first time. She had a history of mild hypertension and occasional fluid retention for several years and had taken diuril (chlorothiazide) occasionally until 1975, when she was placed on hygroton (nonthiazide); however, she was taking no medication at the time she was placed on hydrochlorothiazide on 7 February 1980. There was no past history of allergy or of any previous medication reaction.

Physical examination on admission showed the patient appearing slightly cyanotic, anxious and hyperventilating at 35-40 respirations per minute. Blood pressure was 106/75, pulse 120, temperature 100.4 (*R*). The lungs were initially clear to auscultation; subsequently, however, expiratory rales were noted. There was no neck vein distention or peripheral edema.

An EKG showed sinus tachycardia with two premature ventricular contractions. Blood gases were as follows: PCO_2 22, PO_2 83, HCO_3 15 and pH 7.44. Electrolytes: sodium 136, potassium 3.5, chloride 94, CO_2 24, hemoglobin 16.1, hematocrit 49, WBC 2300 with 16 segs, 29 bands, 1 basophil, 2 metamyelocytes, 2 eosinophils, 35 lymphocytes and 15 monocytes. Blood sugar 138, BUN 13. Admission chest radiographs showed a normal heart size and diffuse interstitial pattern of increased markings in both lung fields with numerous interlobular lymphatic lines bilaterally and a small amount of pleural fluid bilaterally (Fig. 1).

Interstitial pneumonia or an allergic reaction were the primary considerations. She was given furosemide (Lasix®) 20 mgIV and methylprednisolone sodium succinate (Solu-Medrol®) 200 mgIV, which was repeated six hours later, and 20 mg of prednisone the next day. Within 24 hours she was markedly improved clinically with normal pulse and respirations and no cough, wheezing, or shortness of breath. Followup chest radiographs 24 hours following admission showed the lungs completely clear and a small amount of pleural fluid present at both costophrenic angles.

She was discharged 8 February 1980 to be continued on prednisone for three additional days.

Fig. 1. PA Chest film on admission shows diffuse interstitial pattern of increased markings, numerous interlobular lymphatic lines bilaterally and some pleural fluid.

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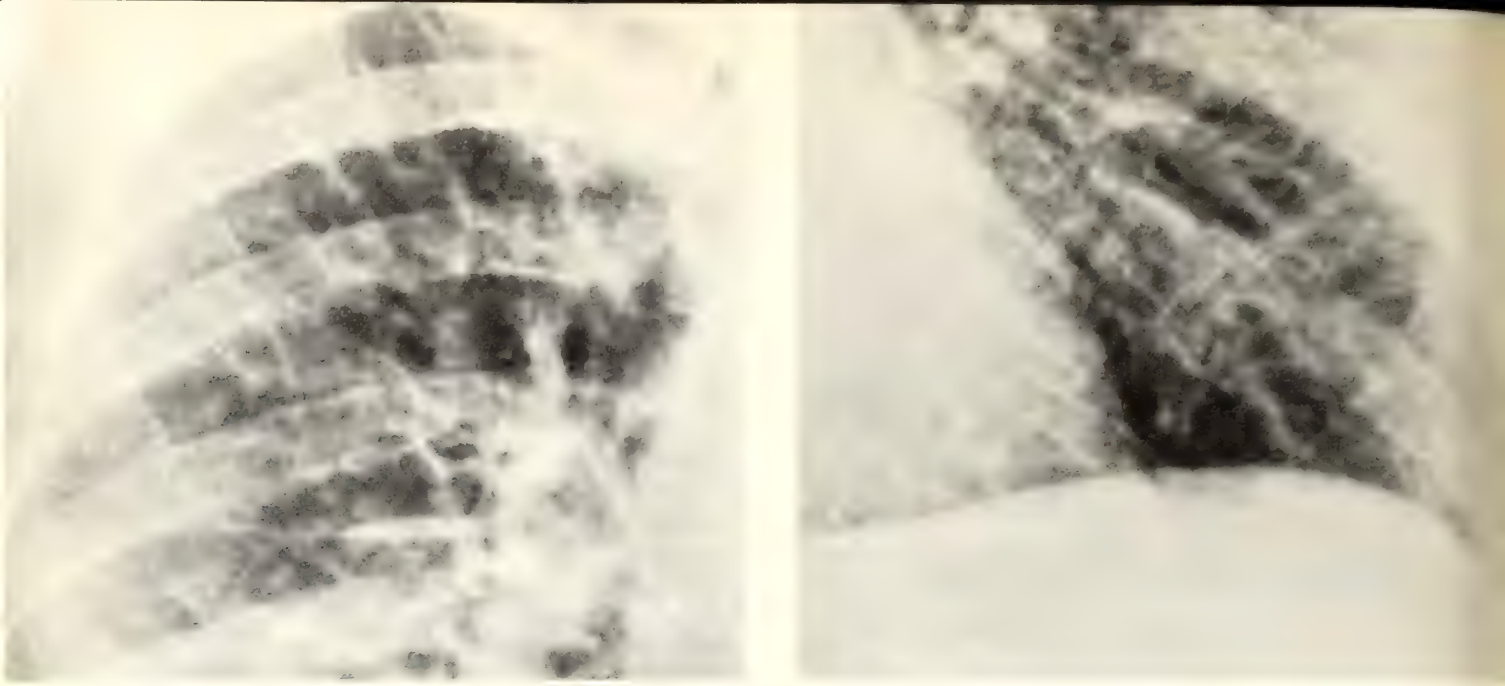

Fig. 2. Close-up views of admission PA chest film showing prominent Kerley's A lines (left), and prominent C lines with relative paucity of B lines (right).

Discussion

In a comprehensive review of drug-induced lung disease published in 1979, Morrison and Goldman refer to only four published cases of pulmonary complications of hydrochlorothiazide.[1] In view of the wide usage of this drug, it appears that pulmonary complications associated with its use are either quite rare, infrequently reported, infrequently recognized as such, or most likely a combination of all three. The four previously published case reports of pulmonary edema secondary to hydrochlorothiazide all showed an onset of symptoms within one hour after ingestion of a single tablet. Acute respiratory distress or chest pain were the predominent complaints in all four patients. Their chest radiographic findings were reported as consisting of alveolar and interstitial infiltrates in one case,[2] interstitial infiltrates in one case,[3] and in the other two cases pulmonary vascular congestion and congestive changes.[4]

In our case there was a diffuse interstitial pattern of increased density in both lung fields with striking prominence of Kerley's A and C lines but with very few B lines (Fig. 2). Pleural fluid was also present bilaterally. Although pulmonary edema from any cause may be responsible for Kerley's lines, Felson states that more than half the patients in whom the A and C lines predominate are suffering from drug sensitivity reactions.[5]

A significant number of patients for whom hydrochlorothiazide is prescribed have some degree of cardiovascular disease and represent a patient group in whom the sudden onset of chest pain and acute respiratory distress on the basis of cardiovascular disease would not be completely unexpected. In this clinical setting, radiographic findings of pulmonary edema might easily be attributed to a primary cardiovascular etiology. Consequently, consideration must be given to the possibility of hydrochlorothiazide sensitivity in any patient with acute pulmonary edema and most particularly in patients with an interstitial type of pattern and a normal cardiac silhouette.

Hydrochlorothiazide is dispensed under these labels: SK-Hydrochlorothiazide,™ Diaqua,™ Lexor,™ Hydromal, Zide, Hydro-Z-50, Thianal, Chlorzide, Diu-Scrip and Hydro-Chlor.

The author thanks Dr. Bryon Cook for permission to use this case, Mrs. Shari Burke for assistance in manuscript preparation, and Ms. Cynthia Shuba for the photographic reproductions.

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Sickle Cell Disease: Acute Splenic Sequestration Crisis

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THE FIRST case of sickle cell anemia in a patient born in the United States was reported in this Journal in 1911 by Dr. R.E. Washburn of the University of Virginia.[1] Today sickle cell anemia (homozygous sickle hemoglobin, or HbS/S), is the most common clinically significant hemoglobinopathy in the United States. Approximately 30% of the mortality associated with sickle cell anemia occurs during the early childhood years.[2] Significant morbidity from the disease and its accompanying infectious complications also occurs during the first three years of life.[3]

A syndrome of sudden death in young children with sickle cell anemia was outlined by Jenkins, Scott and Baird in 1960.[4] More recent reviews have documented that these episodes of sudden death are frequently associated with pneumococcal infection or acute splenic sequestration crises.[5] Pneumococcal infection in infants and children with sickle cell anemia is associated with a 37% mortality rate.[6] Acute splenic sequestration crises may result in a 20% mortality.[7]

We report the case of a child previously not known to have sickle cell anemia who presented with a fatal acute splenic sequestration crisis.

From the Department of Pediatrics, University of Virginia Medical Center, Charlottesville VA 22908. Address correspondence to Dr. Sabio.

Submitted 12-2-82.

Case Report

The patient was a black male infant who was born at term weighing 3680 grams after an uncomplicated pregnancy, labor and delivery to a 26-year-old black female who had had two previously uneventful pregnancies. The neonatal period was uncomplicated. A "sickle cell prep" (not electrophoresis) was done at 2 months of age and was reported normal. Immunizations were given during the first year of life. There were no significant reported illnesses.

At 24 months of age he was seen with complaints of vague abdominal pain and was found to be febrile. His abdominal exam was reported as normal without organomegaly. Symptomatic therapy was prescribed. One week later he presented to the University of Virginia Hospital Emergency Room with persistent abdominal pain. He had no vomiting or diarrhea. On physical exam his height was in the 10th percentile and his weight was in the 5th percentile. The pulse was 150, respiratory rate 36, and temperature was 38.5°C rectally. He was pale, alert, and crying, with abdominal distension. His liver was felt enlarged to 5 cm below the right costal margin and his spleen was felt down to the iliac crest. His hematocrit was 8.5%, and he was transfused with group O Rh negative blood (10 ml/kg).

Minutes after his arrival at the hospital he had a cardiorespiratory arrest. Efforts at resuscitation were unsuccessful. An autopsy showed splenomegaly with a splenic weight of 210 grams. Hemoglobin electrophoresis revealed homozygous HbS. Followup testing revealed that both parents have sickle cell trait and both surviving brothers have a normal hemoglobin electrophoresis with no evidence of sickle hemoglobin.

Discussion

The occurrence of an acute symptomatic episode in a child with a chronic disease is often anticipated, and the presenting signs are evaluated within the context of the underlying chronic disease. The infant described above presented with a fatal complication of his previously undiagnosed underlying disease. Fatal presentations in sickle cell anemia have been documented and occur almost exclusively in childhood. A recent report reviewed seven children, all under 2 years of age, who were not suspected of having sickle cell anemia and who died soon after arrival in the emergency room. The symptoms were nonspecific and not suggestive of sickle cell disease.[8]

It has been suggested that any black child who dies suddenly should have a postmortem blood culture and hemoglobin electrophoresis. Following these guidelines, seven children were identified in the Los Angeles area whose only clinical manifestation of sickle cell anemia was sudden death.[2]

Infection is the leading cause of death in children with sickle cell anemia, followed by acute splenic sequestration and sudden death.[2] Since the major offending organism in fatal infections has been pneumococcus, efforts are made to treat this empirically in the febrile child with known sickle cell anemia and to attempt prevention with the polyvalent pneumococcal vaccine. A prospective study of 22 children identified at birth as having sickle cell anemia or a variant sickle hemoglobinopathy revealed that with consistent comprehensive medical care this group of patients had exhibited no mortality. The incidence of infectious episodes was 38% and there were two episodes of acute splenic sequestration.[9] This remarkably favorable outcome was in contrast to the 31% mortality rate observed when patients were diagnosed at birth and no consistent medical followup was made available.[1] Children who have not been diagnosed as having sickle cell anemia would presumably be at similar risk. The clinical manifestations observed at the time of a fatal complication in these children were not characteristic of sickle cell anemia, emphasizing the need for early clinical suspicion and accurate laboratory diagnosis.

The routine laboratory procedure of choice for the early diagnosis of sickle cell anemia is hemoglobin electrophoresis,[10] which is readily available through public health departments. The use of a sickle cell prep in early infancy should be discouraged since the high concentration of fetal hemoglobin still present could result in a false-negative result.

Increased awareness of the manifestations of sickle cell anemia in infants and of the diagnostic facilities available for early diagnosis could lead to a reduction in the morbidity and mortality of this disease during early childhood.

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VIRGINIA AUTHORS

Extraintestinal Infections Due to Halophilic Vibrios. Carl W. Armstrong, MD, Jeffrey L. Lake, MS, and Grayson B. Miller, Jr., MD, *Richmond.*

To compare the clinical and epidemiologic features, we reviewed the hospital records and interviewed 18 patients with extraintestinal infections caused by three species of halophilic vibrios. *Vibrio vulnificus,* the organism most frequently isolated in Virginia during the six-year study period, caused primary septicemia in three patients. Fifteen patients had soft tissue infections, most of which followed injuries that were contaminated by seawater and mostly caused by *V. vulnificus* and *V. parahaemolyticus*. All infections occurred during the warm months of the year, and in most cases were an occupational or recreational hazard of fishing. South Med J 76:571–574, 1983

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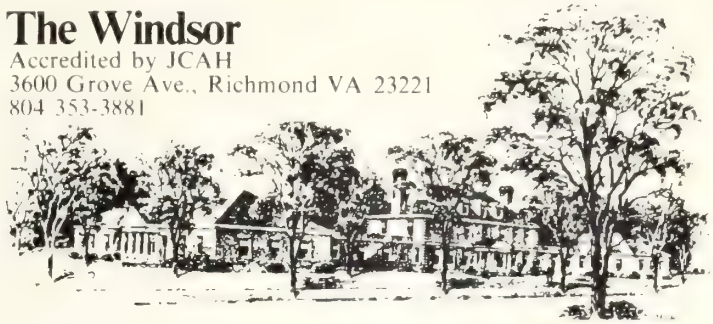

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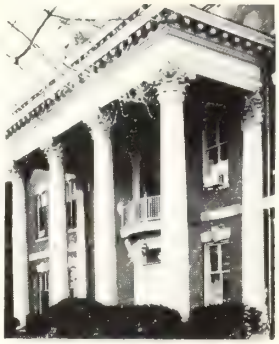

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VIRGINIA MEDICAL

EDITORIAL

A Salute to President Cook

We are indeed fortunate to have Dr. C. Barrie Cook as the new President of The Medical Society of Virginia. He has been an active member of the Society since 1958. He has served this organization energetically and enthusiastically over the years, giving freely and generously of his time in a variety of different areas. This comes as no surprise, for his reputation as a physician and his dedication to our profession are well-known.

It is a special privilege for me to recognize him at this time, due to the fact that our relationship has spanned more than 20 years. Our association has been a close one, allowing me to observe and appreciate first-hand the important characteristics that Barrie will bring to his new position: a high level of professional expertise, a strong sense of commitment, and sound leadership ability.

As the son of a country doctor growing up in rural Virginia, Barrie had envisioned his own future much the same way. The untimely demise of his father created many hardships for his mother, his brother, and him, and he had to overcome many obstacles to reach his goals. He graduated from Hampden-Sydney College with a BS degree in 1945 and went on to George Wahington University, where he received his MD in 1948. As he was preparing for the life of a country doctor, he next accepted a rotating internship at the Medical College of Virginia Hospital from July 1948 to July 1949. He followed this by serving as a medical ward officer at the US Naval Hospital in Charleston, South Carolina, from July 1949 to July 1950 and was a resident in internal medicine at Doctors Hospital of DC from July 1950 to July 1951.

A chance meeting with an old friend of the family resulted in an invitation to visit the Mallory Institute of Pathology in Boston. This proved to be the turning point in his career and he studied there from July 1951 to October 1952. He served in the US Navy from October 1952 to April 1954 and was chief of the Department of Pathology at the US Naval Hospital in Memphis, Tennessee. He returned to Mallory in 1954 and completed his studies in July of 1956. At that time, he returned to the Washington metropolitan area, where he became associated with the Oscar B. Hunter Memorial Laboratory and also joined the pathology staff of the Doctors Hospital of DC.

After three years in the practice of pathology, Dr. Cook was ready to carve his own path. In December 1959 he founded Cook Pathology Laboratory in Fairfax, Virginia. This organization has grown from a handful of faithful employees to one of the largest reference laboratories in the mid-Atlantic states. It is now known as American Medical Laboratories, Inc., a name indicative of the scope of its operation.

In 1961 Barrie established the laboratory operations at the newly opened Fairfax Hospital in Falls Church, Virginia, where he remains to this day as chairman of the Department of Pathology. The Fairfax Hospital has grown to a 650-bed teaching institution of great repute in the 22 intervening years.

The magnitude of these activities alone would have kept the average man more than busy, but Dr. Cook is not your average man. Through the years, he has divided his time and talents among a number of other major undertakings. He was instrumental

in forming the Comprehensive Health Planning Council of Northern Virginia in 1969, he was a member of the Executive Committee of the Fairfax County Medical Society for eight years and also served as its president from 1968 to 1969; he was vice-councilor of The Medical Society of Virginia from 1972 to 1974 and councilor from the 10th District from 1975 to 1980; co-chairman of the Joint Practice Committee of that organization since 1975; and a member of the Governor's Advisory Committee on Medicare and Medicaid since 1978. Dr. Cook has been a member of the American Society of Clinical Pathologists since 1958, serving on the Council on Radioisotopes from 1958 to 1960. He also was a member of the Board of Censors from 1975 to 1976 and again in 1982 and has been an at-large member of the Finance Committee and Secretary of the Advisory Council since 1982.

Dr. Cook has been active in the Boy Scouts of America, serving as a committee member from 1964 through 1973 and as committee chairman from 1968 to 1973. He has served as chairman of the Fairfax Symphony Orchestra Development Committee from 1979 to the present, devoting many hours of his personal time to fund-raising activities. He is also a member of the Wolf Trap Farm Park for the Performing Arts, a member of the Wolf Trappers and has served as chairman of their fund-raising campaigns.

Dr. Cook has also been very active in the Hampden-Sydney College Alumni Association, serving as vice-president from 1978 to 1979 and president from 1979 to 1980. He currently serves on the boards of the Northern Virginia Regional Park Authority, the Fairfax Rotary Club, the Fairfax Symphony Orchestra and the George Mason Bank.

Barrie was married to Jean Carrington of Richmond in 1949 and together they have raised two sons and three daughters. Jean is an accomplished concert pianist who has performed all over the world and has been featured on our local radio stations. Barrie enjoys golfing with his friends in his leisure time and becomes an avid skiing fan at the flutter of the first snowflake.

Barrie has earned the respect of those of us who have worked with him professionally and many of those who have served with him in his busy life. The Society is fortunate that he has been selected as its leader in these difficult days. We can rest assured that he will lead us and represent us well throughout the year.

IRA D. GODWIN, MD

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United We Stand

THERE ARE LEADERS of medicine in Virginia whose names are household words, at least among the medical profession—the President and the councilors of The Medical Society of Virginia, the delegates to the American Medical Association among others. These stand out from the crowd, but what about the foot soldier of medicine, the practitioner who cares for his/her patients with love and affection, as well as the expected medical expertise. This physician often pays little attention to the organizational aspects of medicine; he/she is happy in the daily routine of practice or teaching.

No criticism is to be leveled toward this physician; each of us is an individual and our interests vary. But wouldn't it be wonderful if this physician, often equipped with superior and diverse talents, could be persuaded to lend a little of his/her time and effort to the organized promotion of our common cause, establishment of the best possible medical care for the people of Virginia.

The physician today is faced with the problem of the acronyms, DRG, HMO and PPO among others. Concerted effort within our organization, The Medical Society of Virginia, will enable us to promote a solution that will not only be beneficial to the medical profession, but best of all will provide superior medical care to our patients.

How can this be accomplished? How can the individual physician be convinced that united we stand, divided we fall? The United States government and the other third-party payers have capitalized on our lack of cohesion; they recognize that medicine does not always provide a united front. The American Medical Association and The Medical Society of Virginia provide excellent leadership, but both organizations need the interest and help of every single physician. As a starter, why don't you call your own Medical Society of Virginia councilor today; offer your services in this all-out war that well may be the Armageddon of medicine as we know it today.

EDWIN L. KENDIG, JR., MD

VIRGINIA MEDICAL OBITUARY

T. Henry Dickerson, MD

Dr. Thomas Henry Dickerson, for 35 years medical supervisor at the Martinsville DuPont company, died August 11 at North Carolina Baptist Hospital, Winston-Salem. He was 72 years old.

A native of Wilmington, Delaware, Dr. Dickerson was educated at Harvard University and the University of Delaware, and received his medical degree in 1937 from Thomas Jefferson University, Philadelphia. He came to Martinsville in 1941 and there served in the post of medical supervisor for DuPont until his retirement in 1976.

A past president of the Patrick Henry Medical Society, Dr. Dickerson had been a member of The Medical Society of Virginia since 1944. He was also a member of the American Occupational Medical Association and the Industrial Medical Association.

Memoir of Thomas L. Lanier, Jr. 1938-1983

By Harold L. Williams, MD

Dr. Thomas L. Lanier., Jr., age 45, of Newport News, died on August 20, 1983.

He was a lifelong resident of Newport News and a graduate of Warwick High School where he starred in basketball. He later graduated from Dartmouth College and the University of Virginia Medical School. Dr. Lanier interned at Hartford General Hospital and completed his residency at Grady Memorial Hospital, Atlanta, and the Medical University of South Carolina. He continued his specialty training at the Lahey Clinic and Methodist Hospital, Houston. He was a diplomate of The American Board of Colon & Rectal Surgery, and had practiced general and vascular surgery at Riverside Hospital, Newport News, for the past ten years. He served his country for two years, 1968-1970, in Vietnam as a surgeon near the front line.

Our heartfelt sympathy goes out to his parents, Mr. and Mrs. Thomas L. Lanier and his brother, Robert F. Lanier, all of Newport News, and to his twin sister, Mrs. Nancy Lanier Player of Florence, South Carolina.

Dr. Lanier was a compassionate, kind and considerate man. He was never too busy or too tired to stop and chat about almost anything, anytime. He was incapable of insincerity and detested pettiness. He could no more understand the constant bickering within a hospital staff than he could the mysteries of the cosmos—yet he endeavored to solve both.

He looked for the good in everyone and usually found it. He was slow to criticize and quick to defend his colleagues when they were impugned. He met criticism with self-assessment, adversity with renewed commitment, malice with forgiveness, and hypocrisy with truth.

More than most, Tommy understood humankind and his frailties. He once offered the opinion that cats and dogs were often more humane than humans.

Tommy liked sports, literature, nature and the world around him. Some of us often traded quotes and trivia in the surgical lounge. Tommy could recite every word of Wordsworth's "I Wandered Lonely as a Cloud" and make you feel as if you were *there* with the daffodils "fluttering and dancing in the breeze". Perhaps it was because he *had been there*—to the Lake District of Northern England—and seen the daffodils along the margin of Ullswater "nodding their golden heads". He was never content with a picture or description of something; he wanted to see it. He visited New Zealand, climbed the "Milford Tract" and saw the Sutherland Falls up close.

Tommy seemed like a man in a hurry; in a hurry to experience all that was available in this world. One of the quotes he liked most was by Sir Walter Scott: "One crowded hour of glorious like is worth an age without a name".

Dr. Tommy Lanier, in his way, lived his short life to the fullest. He will be sorely missed by his patients and all those who knew and loved him.

Perhaps Shakespeare said it best:

His life was gentle; and the elements
so mix'd in him that Nature might stand up
and say to all the world 'this was a man!'

WHO'S WHO

The last full-time doctor on Tangier Island, out in the middle of the Chesapeake Bay, died in 1969, and the island's 800 residents have depended recently on twice-weekly visits from doctors flying in from the Eastern Shore.

But on a sunny Saturday in September, a group of doctors from the Tidewater area descended on Tangier, bringing with them a dentist, podiatrist, respiratory therapist, pharmacist, nurses and nurse practitioners, and a number of medical and dental students. It was Tangier's Health Day '83. Every islander was treated to free exams, advice and medication when needed.

Medical prevention was the aim of **Dr. Juan M. Montero**, Chesapeake surgeon, who was a motivating force behind the fair. He and his associates have been helping with an annual Eastern Shore health fair since 1979, and when some Tangier islanders registered at the one last April, Dr. Montero came up with the idea for the Tangier fair.

The project in final form was the work of many persons and organizations. The yearly Eastern Shore health fairs are sponsored by Eastern Shore Rural Health, Inc., a federally funded effort dedicated to expanding health care services, and its staff coordinated the Tangier event, underwrote the expenses, and provided personnel. Tangier's Rudy Thomas donated ferry passage to the island from Onancock. Printed materials and medical supplies were provided by the American Cancer Society, National Arthritis Foundation and American Heart Association.

The visiting health team set up stations with signs describing the medical service available at each. Lines of islanders moved from station to station, and 179 of the island's residents registered for some type of activity, including 58 of the island's 80 grandchildren. At the close of the day, the islanders treated all the visitors to a seafood dinner in the community's recreation center. And after it was all over, Joe Fahy of the *Virginian-Pilot* wrote a front-page feature story, from which this information was taken.

Besides Dr. Montero, these other Medical Society of Virginia members were in the contingent that journeyed to Tangier: **Dr. Teofilo C. Mascarinas, Jr.,** *Virginia Beach* internist; **Dr. Gloria A. Santiago,** gynecologist in *Virginia Beach*; and **Dr. Jose E. Gonzales,** *Chesapeake* urologist.

Dr. Montero was recently elected president of the Chesapeake Medical Society. Chosen to serve with him were **Dr. William P. Brown,** secretary, and **Dr. Alfredo Soriano,** treasurer.

Dr. Alex P. Yadao, whose *Northern Virginia* practice is based in Triangle, has been inducted as president of the American College of International Physicians. He has been a trustee of the college since 1980 and has served on many of the organization's committees.

President-elect of the Society for Surgery of the Alimentary Tract is **Dr. R. Scott Jones,** chairman of the Department of Surgery at the University of Virginia School of Medicine. The 1,000-member organization focuses its activities on surgical problems of the digestive system.

New vice-speaker of the American College of Radiology is **Dr. Karl K. Wallace, Jr.,** *Virginia Beach*, who from 1976 to 1980 was Speaker of the Medical Society of Virginia's House of Delegates.

Tangier treat: Dr. Montero with islander Henry A. Daley.
Photograph by Bill Ballenberg

Dr. Robert Fry has been appointed chief physician for the Virginia Department of Corrections. A native of *Portsmouth*, Dr. Fry is a Medical College of Virginia graduate, has conducted a private practice in Suffolk, and has been institutional physician at the Deerfield Correctional Center.

New president of the Mid-Atlantic Group Psychotherapy Society is **Dr. Bernard M. Williams,** Charlottesville, who took office during the Society's spring meeting in Wintergreen. Dr. Williams is also president elect of the Neuropsychiatric Society of Virginia.

Dr. Vamik D. Volkan, Charlottesville, took office as president of the International Society of Political Psychology at its scientific conference last summer in Oxford, England. He is the first medical professional to lead the 1,000-member organization, which strives to facilitate improved communications on international affairs across the boundaries of disciplines, geography and politics; its members also exchange research on such topics as the performance of leaders under stress.

A psychoanalyst, Dr. Volkan received his MD degree at the University of Ankara Medical School in Turkey. He joined the University of Virginia faculty in 1963 and now is medical director of the university's Blue Ridge Hospital. He is president of the Virginia Psychoanalytic Society.

To **Dr. Joshua Price Sutherland,** Grundy, went a coveted award when Emory and Henry College held its 1983 commencement exercises. It was the William and Martha DeFrience Award, given annually by the college's board of trustees to an alumnus or faculty member who has made "distinctive contributions to civilization." Dr. Sutherland went on from Emory and Henry to the University of Alabama, then earned his medical degree at the University of Virginia. For 15 years he worked alongside his father, Dr. Tivis C. Sutherland, serving remote areas of Dickenson and Buchanan Counties.

In 1963 Dr. Sutherland opened a medical clinic in Grundy and today serves the clinic's entire patient load, assisted by a staff that includes his wife, Nell, and a daughter, Betty. In addition, he is chairman of the board of trustees of Buchanan County General Hospital, which he was instrumental in founding.

Dr. William D. Mayer, Norfolk, has been reelected to a second term as chairman of the board of regents of the National Library of Medicine, which is a division of the National Institutes of Health and the major repository of the world's medical and biomedical science literature. Dr. Mayer, a pathologist, is president of the Eastern Virginia Medical Authority.

Dr. John Boniface, Jr., Galax, has been appointed to a four-year term on the Grayson County School Board.

After 55 years, the exploits as a high school and college athlete of **Dr. Lemuel E. Mayo, Jr.,** have been recognized with a special citation from the Virginia Sports Hall of Fame. Now 71 years old, the Portsmouth surgeon led Wilson High School to the state basketball championship in 1928 and in the same year was the leading scorer in the state track meet. At the University of Virginia, where he finished medical school at the age of 20, he was captain of the Cavaliers. Today golf is Dr. Mayo's sport; he once played to a five handicap, but says he's now happy to break 90.

Two Medical Society of Virginia members were named fellows of the American College of Radiology during its recent meeting in Chicago. They are **Dr. Barry T. Katzen,** *Falls Church*, and **Dr. Donald J. Kenneweg,** *Fredericksburg*.

Dr. Nellie Martin Sankaran, *Suffolk*, has been elected to fellowship in the American Academy of Pediatrics.

Dr. Edward W. Hook, chairman of the Department of Internal Medicine at the University of Virginia, has been elected to the Institute of Medicine of the National Academy of Sciences.

When **Dr. Charles F. Gaylord** of *Staunton* started out in medicine almost 50 years ago, house calls were an integral part of family practice, and when he retired last summer, at the age of 73, he was still visiting homebound patients.

"Everyone made house calls back then," he reminisced for reporter Debbie Meade of the Staunton *Leader*. "Most deliveries were out of homes." And, he remembered, it was common to see three generations in a single home—sometimes even four or five generations. The demise of the extended-family home is hard on older people, he observed. "That's why the need for nursing homes is so much greater today."

Born in *Connecticut*, **Dr. Gaylord** moved to Cleveland as a teenager and took his undergraduate and medical degrees at Western Reserve University in Cleveland. He trained at a Cincinnati Hospital, then he and his wife moved to Staunton. They have lived there ever since, rearing a daughter and a son.

What will Dr. Gaylord do now, without office and house calls? Work around the house and yard, he told the reporter. "No schedule," he said, smiling. "It's very nice."

VIRGINIA MEDICAL CLASSIFIED

Virginia Medical classified ads accepted at the discretion of the Editor. Rates to Medical Society of Virginia members: $15 per insertion up to 50 words, 25¢ each additional word. To non-members: $30 per insertion up to 50 words, 25¢ each additional word. Deadline: 5th day of month prior to month of publication. Send to the Advertising Manager, 4205 Dover Road, Richmond VA 23221, (804) 353-2721.

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There are many excellent handbooks of effective writing, among them *The Elements of Style*, by William Strunk, Jr., and E. B. White (MacMillan); *The Careful Writer: A Modern Guide to English Usage*, by Theodore M. Bernstein (Atheneum); and *How to Write and Publish a Scientific Paper*, by Robert A. Day (ISI Press).

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b—book review	*—medical article
cr—committee report	m—memoir
ed—editorial	nb—news bureau
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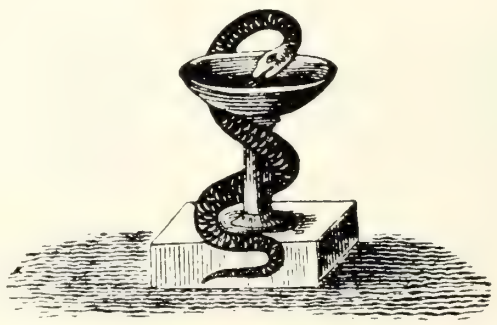

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- Studied extensively in the sleep laboratory—the most valid environment for measuring hypnotic efficacy.[1-12]
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- Contraindicated in patients who are pregnant or hypersensitive to flurazepam.
- Caution patients about drinking alcohol, driving or operating hazardous machinery during therapy.

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Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation

Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

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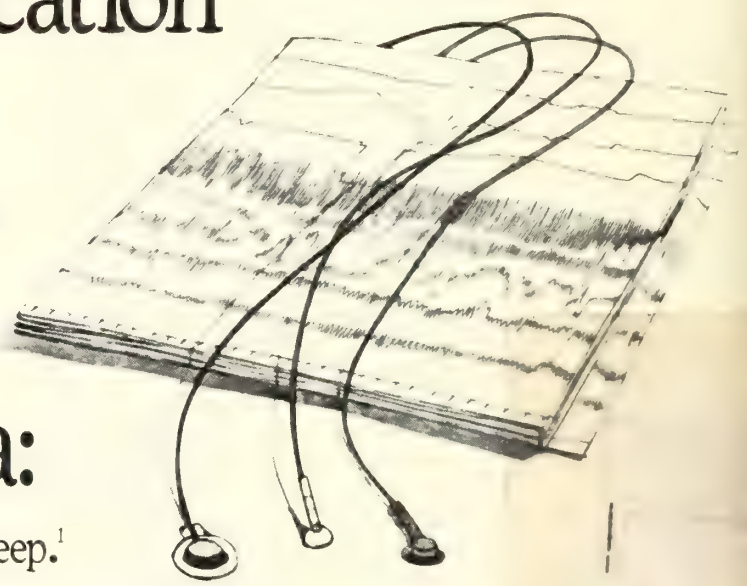
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